National Library of Canada
Canadian Theses Service
Ottawa, Canada
K1A 0N4

Bibliothèque nationale du Canada
Service des thèses canadiennes

NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Reproduction in full or in part of this microform is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30, and subsequent amendments.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

La reproduction, même partielle, de cette microforme est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30, et ses amendements subséquents.

NL-339 (r. 88/04) c

Canada

National Library of Canada
Bibliothèque nationale du Canada

Canadian Theses Service
Service des thèses canadiennes

Ottawa, Canada
K1A 0N4

The author has granted an irrevocable non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of his/her thesis by any means and in any form or format, making this thesis available to interested persons.

L'auteur a accordé une licence irrévocable et non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de sa thèse de quelque manière et sous quelque forme que ce soit pour mettre des exemplaires de cette thèse à la disposition des personnes intéressées.

The author retains ownership of the copyright in his/her thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without his/her permission.

L'auteur conserve la propriété du droit d'auteur qui protège sa thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

ISBN 0-315-55570-X

Canadä

THE UNIVERSITY OF ALBERTA

THE EFFECTS OF ENDURANCE TRAINING ON PHYSIOLOGICAL, NUTRITIONAL AND ENDOCRINE VARIABLES IN MEN AND WOMEN

BY

GARRY D. WHEELER MSc.

A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE
OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF PHYSICAL EDUCATION AND SPORTS STUDIES

EDMONTON, ALBERTA

(FALL, 1989)

THE UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR: GARRY D. WHEELER

TITLE OF THESIS: THE EFFECTS OF ENDURANCE TRAINING ON PHYSIOLOGICAL, NUTRITIONAL AND ENDOCRINE VARIABLES IN MEN AND WOMEN

YEAR THIS DEGREE WAS GRANTED: 1989

Permission is hereby granted to the UNIVERSITY OF ALBERTA LIBRARY to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific research purposes only.

The author reserves other publication rights, and neither the thesis nor extensive abstracts from it may be printed or otherwise reproduced without the author's written permission.

(Student)

19052 72 B
AVE, EDMONTON,
T5T 5T1

(Address)

Date: 1989

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the faculty of Graduate Studies and research for acceptance, a thesis entitled: **The effects of endurance training on phsysiological, nutritional and endocrine variables in men and women.**

Submitted by: Garry D. Wheeler

in partial fulfillment of the requirements for the degree of:

Doctor of Philosophy.

....
(Supervisor)

....

W. David Pierce
....

....

Murray W. Smith
....

W F Epling
....

Date:

Abstract.

The purposes of the investigation were (1) To examine the effects of endurance training on VO_2 max., body fat and lean body mass. (2) to examine a model of Activity Anorexia; specifically appetite suppression and the effects of weekly rate of change of activity on food intake, and (3) to investigate the role of dietary intake and energy balance in alterations in reproductive hormones and LH pulse frequency in high mileage and beginner runners.

Three groups of volunteer subjects were selected. Ten high mileage runners (HMRM) and 10 female runners along with 15 male (CONM) and 15 female (CONF) sedentary subjects as a control group. In addition a third group of 20 sedentary men (TRM) and 20 women (TRF) was selected to take part in a six month running training program designed to increase training distance to approximately 40-56 km/week. HMRM, HMRF, CONF and CONM subjects were required to pursue their normal training routine or sedentary lifestyles and to record dietary intake for six months.

All subjects underwent pre, mid and post training program assessment of aerobic capacity and body fat and food intake based on three day diet diaries. Blood samples were collected from HMRM and TRM to determine serum hormone levels and parameters of LH pulsatile release. Ten HMRM and 7 HMRF; 14 TRM and 13 TRF and 7 CONM and 11 CONF subjects completed the investigation.

Six months of endurance running training resulted in a significant improvement in VO_2 max. and decreased percent body fat in TR. Results indicated no differences in caloric intake or diet composition either before or after training among the groups. Trend analysis revealed that a subset of the TRM exhibited a significant quadratic and quartic trend in response to the training program. This was characterised by a drop in food intake as training began. The drop in caloric intake was associated with the rate of increase in weekly running. TRF exhibited no distinctive dietary trend as a result of training.

There was a significant pre-training difference in total testosterone between HMRM and TRM. Training resulted in a significant decrease in total testosterone in the TRM group. There was no difference in LH pulsatile release either before or after training. Correlation analysis revealed that total testosterone was directly related to dietary intake factors in HMRM but not sedentary men at the pre test. Following the training program total testosterone was directly related to dietary intake in TRM and HMRM as a group.

Trend analysis suggests that exercise had a suppressive effect on appetite in subjects with a higher weekly rate of increase in running. In male runners alterations in total testosterone would appear to be directly related to nutrient intake and not LH pulsatile release. This may represent a metabolic adaptation to a negative energy balance.

Acknowledgement:

This study was supported in part by a research grant from the Central Research Fund, the University of Alberta, Edmonton, Canada.

Thesis Contents

Chapter	Section#	Chapter Title	Page Number
Chapter 1		Introduction and statement of the problem	1
Chapter 2		Methodology	32
Chapter 3		Results	58
	section 1	Group personal data and subject compliance and attrition.	59
	section 2	Training achievements and the effects of training on VO2 max.	62
	section 3	The effects of training on anthropometric variables: Body fat, body weight and lean body mass.	74
	section 4	The effects of endurance training on caloric intake and dietary composition of HMR, TR and CON.	86
		Individual dietary profiles among TRM and TRF	105
		Energy expenditure and caloric intake (energy balance)	121
		Comparison of the nutrient intake of HMR, TR and CON to the RNI for Canadians.	128
		Effects of a six month training program on Eating Attitudes and Eating Disorder Inventory Scores.	137
	section 5	The effects of six months of endurance training on total testosterone, SHBG, free androgen index,	144

cortisol, FSH, LH, PRL, and LH pulsatile release in HMRM and TRM.

section 6 Correlations between total testosterone and nutritional variables 160

Chapter 4 Discussion of Results 169

Part1 Effects of exercise on measures of aerobic capacity and percent body fat 169

Part2 An examination of the results as they support a model of Activity Anorexia. 170

Chapter 5 Conclusions and Recommendations 204

References 209

Appendices

Thesis Contents: List of Tables.

Table Number.	Title of Table	Page Number
Table1.	Subject Profiles.	61
Table 2.	Training achievements: HMR and TR.	64
Table 3.	The effects of training on VO2 max.	70
Table 4.	Effects of training on body weight: Means and SEM.	76
Table 5.	Percent body fat in HMR, TR and CON subjects.	81
Table 6.	Mean lean body mass in HMR, TR and CON subjects.	85
Table 7.	Effects of training on caloric intake.	88
Table 8.	Carbohydrate intake in HMR, TR and CON (gms).	92
Table 9.	Dietary fat intake (gms).	97
Table 10.	Dietary protein intake (gms).	102
Table 11.	Dietary surplus/deficit (kilocalories) in HMR, TR and CON.	123
Table 12.	Dietary deficit/surplus ratios before and after training.	127
Table 13.	Comparison of caloric intake; Pre measure to Canadian Dietary Standards.	130
Table 14.	A comparison of dietary protein intake in HMR, TR and CON to Canadian Dietary Standards (pre program measures).	131
Table 15.	Caloric intake in HMR, TR and CON following six months of endurance training: Comparison to RNI for Canadians.	133
Table 16.	Protein intake following a six month endurance training programme: Comparison' to Canadian RNI.	134
Table 17.	Carbohydrate intake as a percentage of total nutrient intake.	135
Table 18.	Dietary fat intake as a percentage of total nutrient intake.	136

Table 19. EAT and EDI scores in HMR, TR and CON before and after training. 137

Table 20. Hormonal changes during a six month training program. 146

Table 21. Summary of statistics: HMR v TRM LH pulse parameters. 156

Table 22. Summary of T test results: LH pulse parameters before and after training in TRM. 158

Table 23. Correlation coefficients for HMR: Pre and Post Investigation v Pre and Post T levels. 161

Table 24. Correlations: Pre and Post training: TR group. Selected variables v testosterone. 162

Table 25. Correlation Coefficients: Grouped data (HMR and TR): Pre and Post training v Testosterone. 163

Table 26. Factor analysis: Pre measures: Orthoganal transformation: varimax. 166

Table 27. Factor analysis: Post measures: Orthoganal solution-varimax. 167

Table 28. Factor analysis: Post measures: Oblique solution: Primary pattern. 168

Table 29. The effects of exercise and opiate antagonists on LH pulsatile release in men. 186

Table 30. A comparison of investigation conditions of gonadotropin secretion in trained men and control subjects. 190

Table 30. Standardized pulse frequencies in exercise investigations. 194

Thesis Contents: List of Illustrations.

Figure/Graph	Figure Title	Page Nos.
Figure 1.	Distances achieved:HMR, TR and CON by group.	65
Figure 2.	Training distances achieved: HMR and TR: Group*gender.	66
Figure 3.	The effects of training on VO2 max by group.	71
Figure 4.	The effects of training on VO2 max: group* gender (men).	72
Figure 5.	The effects of training on VO2 max: group* gender (women).	73
Figure 6.	Effects of training by group (body weight).	77
Figure 7.	Effects of training on body weight: group*gender (men).	78
Figure 8.	Effects of training on body fat by group.	82
Figure 9.	Group differences in body fat in women subjects.	83
Figure 10.	Effects of training on caloric intake by group.	89
Figure 11.	Gender differences in caloric intake (covariate = lean body mass).	90
Figure 12.	Effects of training on carbohydrate intake in HMR, TR and CON.	93
Figure 13a.	Effects of training on carbohydrate intake in HMR, TR and CON (adjusted means: LBM).	94
Figure 13b.	Effects of training on carbohydrate intake in HMR, TR and CON (adjusted means: Weight).	95
Figure 14.	Effects of training on the dietary fat intake in HMR, TR and CON.	98
Figure 15a.	Effects of training on dietary fat intake: adjusted means by gender(cov = LBM).	99

Figure 15b. Effects of training on dietary fat intake adjusted means by gender (cov = Weight). 100

Figure 16. Effects of training on dietary protein intake in HMR, TR and CON. 103

Figure 17. The effects of training on dietary protein intake by gender (adjusted means). 104

Figure 18. Mean caloric intake in 10 HMRM during a six month training program. 106

Figure 19. Mean caloric intake in 9 HMRF during a six month training program. 106

Figures 18a - j Individual dietary and training profiles (HMRM). 111

Figures 19a - i Individual dietary and training profiles (HMRF). 116

Figure 20. Dietary and training profiles for 5 TRM responders. 110

Figure 21. Dietary and training profiles for 5 TRM non-responders. 110

Figure 22. Dietary surplus/deficit in HMR, TR and CON before and after training. 124

Figure 23. Dietary surplus/deficit in HMRM, TRM and CONM. 125

Figure 24. Dietary surplus/deficit in HMRF, TRF and CONF. 126

Figure 25. EAT scores by group before and after training. 140

Figure 26. EDI scores by group before and after training. 141

Figure 27. EAT scores: Group*gender (women) 142

Figure 28. EDI scores: Group*gender (women) 143

Figure 29. Effects of training on total Testosterone in HMR and TR subjects. 147

Figure 30. Effects of training on SHBG capacity in HMR and TR subjects. 148

Figure 31. Effects of training on the Free Androgen Index in HMR and TR subjects. 149

Figure 32. Effects of training on FSH levels in HMR and TR subjects. 150

Figure 33. Effects of training on Cortisol in HMR and TR subjects. 151

Figure 34. Baseline LH levels in HMR and TR. 152

Figure 35. Effects of training on LH levels in TR subjects. 153

Figure 36. Effects of training on Prolactin in HMR and TR subjects. 154

Figure 37. Mean LH pulse frequency in HMRM and TRM. 157

Figure 38. Mean LH Pulse frequency in TRM before and after six months of endurance training. 159

Figure 39. A hypothetical model of the development of An Activity Anorexia. 208

Appendices:

Appendix 1:	Raw data: All groups.
Appendix 2a:	Means, standard deviations and standard error for all variables by group: High Mileage runners.
Appendix 2b:	Mean data by group: Training Group.
Appendix 3:	Raw data: nutrient intake: relative to body weight.
Appendix 4:	Nutrient intake (TRM and TRF) (10 TRM and 9 TRF)
Appendix 5:	Luteinizing hormone data: Six hour sampling: High mileage runners.
Appendix 5b:	Luteinizing hormone data: Trainee; Pre and Post.
Appendix 5c:	Luteinizing hormone raw data: (all groups).
Appendix 6a:	Correlations: HMRM Pre data: Total T v Nutrient intake.
Appendix 6b:	Correlations: HMRM post data: Total T v Nutrient intake.
Appendix 6c:	Correlations: TRM Pre data: Total T v Nutrient intake.
Appendix 6d:	Correlations: TRM Post data: Total T v Nutrient intake.
Appendix 7a:	Correlations: Group Data Pre.
Appendix 7b:	Correlations: Group Data Post.
Appendix 8:	Nutrient composition of HMR, TR and CON before, during and after a six month training study.
Appendix 9:	Three day diet diary.
Appendix 10:	Statistics Summary Tables: (1 - 32).

Chapter 1.

Introduction and Statement of the Problem:

Introduction:

Running has become an important part of the lives of millions of individuals either as a mode of training for fitness, relaxation and weight control. Although many benefits have been attributed to involvement in running programs recently less desirable effects have given rise to concern. Runners have been described as "neurotic," "negatively addicted" and "anorectic" although traditionally the role of activity has been disregarded as an etiological factor in the development of eating disorders.

However, recent evidence supports a role of activity in alterations of eating behaviour under exercise conditions. A model of *activity anorexia* has been proposed in animals under conditions of food deprivation and opportunity to exercise which may generalize to the human species (Epling and Pierce, 1988). Alterations in dietary behaviour associated with exercise may have other physiological consequences.

Alterations in the reproductive axis in women and men, including amenorrhea and decreased circulating total and free testosterone may in part be due to alterations in caloric intake and/or energy balance.

The purpose of the investigation was to investigate the effect of opportunity to exercise (via a 6 month training program) on caloric intake in men and women as it relates to a model of activity anorexia in animals and to examine the role of caloric intake and energy balance in changes in the hypothalamic-pituitary-gonadal axis associated with endurance training programs.

A Review of the Literature:

There are many physiological and psychological benefits associated with programs of endurance running training. Progressive training programs have resulted in significant improvements in cardiovascular function (Clausen, 1977), increases of 15-20% in aerobic power (Saltin, 1968; Roskam, 1967; Ekblom, 1968; Kasch, 1973; Pollock, 1973; Saltin et al., 1980) and decreased percent body fat (Depres et al., 1985; Pavlou et al., 1985; Thomas, Adeniran, Etheridge, 1984). Psychological benefits include, running as a treatment for depression (Greist et al., 1978; 1979; Blue, 1979; Brown, 1978) and in programs of stress management and anxiety reduction (Blue, 1979; Lion, 1978). Running has been considered as a viable alternative to stress inoculation in stress management programs (Long, 1984;1985).

In contrast some authors have suggested that chronic involvement in distance running may result in anatomical, physiological and psychological alterations which have implications for the health of the athlete.

There has been a large increase in the incidence of chronic overuse syndrome among distance runners (Stanish, 1984; Clement, 1981). Severe disruption of muscle cell structure has been reported in high mileage runners (Hikida et al., 1983; Norregard-Hansen et al., 1982). The habitual or obligatory long distance runner may develop an addiction to running and is unable to stop even when injured. Thus the high mileage runner has been described as "negatively addicted" (Morgan, 1979). Neurotic illness in fitness fanatics forced to cease exercise due to injury has been reported by Little (1981). Recently alterations in hypothalamic-pituitary-gonadal (HPG) function in men and women runners have been documented.

Long distance running has been associated with amenorrhea (Feicht et al., 1978; Frisch et al., 1981; Malina et al., 1978; 1983; Schwartz et al. 1981), abbreviated luteal phase (Shangold et al., 1981; Bonen et al., 1981; Prior et al., 1982), anovulatory cycles (Prior et al., 1982) and delayed menarche (Malina et al., 1978;1983). Chronic training in women has also been associated with bone calcium loss (Nelson et al. 1986; Warren, Brooks-Gunn, 1986). In men chronic running training has been associated with reductions in circulating total and free testosterone levels (Wheeler et al., 1984; Strauss et al., 1985; Ayers et al., 1986; Hackney et al., 1988).

To date the mechanisms associated with alterations in the hypothalamic-pituitary-gonadal axis in men and women runners have not been determined although various hypotheses have been proposed. Alterations in the HPG axis in women runners may be associated with a multi-factorial process including: training intensity and volume, body weight, body fat, lean body mass to weight ratio, alterations in the GnRH (LHRH) pulse generator (with concomitant alterations in LH pulsatile release) and nutrient intake (Cumming and Rebar, 1985).

Alterations in pulsatile LH release have been demonstrated in normally cycling runners under acute exercise and chronic training conditions (Cumming et al., 1985a & b) although the mechanism of suppression is not clear at this time.

It has been suggested that nutrient intake and/or energy balance may be associated with alterations in the HPG axis in women runners (Schwartz et al., 1981; Frisch et al., 1981; Cumming and Rebar, 1985).

A frequent omission in research on athletic amenorrhea is the accurate measurement of dietary intake, although dietary changes frequently accompany exercise programs (Cumming and Rebar, 1983). This is supported by reports of anorectic eating attitudes (Henry, 1982), pathogenic weight control behaviour in women runners (Rosen et al. 1986), dietary changes in runners beginning training pre-menarchally (Frisch et al., 1981) and significantly lower protein intake in the diet of amenorrheic and normally menstruating runners (Schwartz et al., 1981; Plante and Houston, 1974). Evidence also suggests that the cyclical variation of steroid and progesterone levels during the menstrual cycle may affect food intake independent of exercise (Cjaja, 1975). Others have reported low caloric intake in athletes compared to recommended nutrient intake values for training athletes (Short and Short, 1983; Drinkwater et al., 1984; Marcus et al., 1985; Nelson et al., 1986).

Alterations in dietary intake during the menstrual cycle may be mediated by altering steroid levels. Dalvit (1981) reported that caloric intake was 500 kilocalories per day higher during the 10 day post ovulatory period than during a 10 day pre-ovulatory period. She suggested that alterations in progesterone levels during the menstrual cycle may mediate appetite changes. Adding support to this contention Cjaja (1975) found that food intake was suppressed when estradiol levels peaked in female primates.

In contrast, Pirke et al. (1985) reported effects of diet on menstruation. A significant reduction in caloric intake disrupted the menstrual cycle in 60% of a sample of normally menstruating women. Regular cycles resumed six months following resumption of a normal diet. Starvation for 2.5 weeks produced an immature LH secretory pattern in 3 out of 5 normally menstruating women (Fichter and Pirke (1984). Drinkwater et al. (1984) measured caloric intake in a group of 28 women distance runners training 33.4 miles per week, half of whom were amenorrheic. Using the 3 day diet diary recording technique, caloric intake values of 1,750 kcals/day were reported. The level of caloric intake was significantly lower than that recorded in non-exercising women of normal weight (Liebell and Hirsch, 1984) and considerably below levels indicated for women at this activity level. Bates et al. (1982) reported that of 29 women with unexplained infertility, 19 conceived spontaneously on regaining 95% of normal body weight by increasing caloric intake. Others have failed to observe any differences in caloric intake between runners and non-runners despite a significantly lower menstrual frequency in the running group (Dale and Goldberg, 1979).

Various mechanisms have been suggested to explain alterations in testosterone levels reported in male long distance runners. Putative mechanisms include, increased cortisol levels (Cumming, Quigley and Yen, 1983), decreased prolactin levels, increased metabolic clearance or decreased testicular production of testosterone (Wheeler et al., 1984). Others have considered the role of nutrient intake and/ or energy balance (Ayers et al., 1986; Strauss et al., 1985) although no attempt has been made to precisely monitor caloric intake and correlate levels with total and free testosterone in high mileage runners. Nontheless, Strauss et al. (1985) reported significantly reduced total and free testosterone levels in wrestlers at peak season compared to off season levels. The authors considered that low caloric intake contributed to the reduction in circulating levels since low body fat and weight was correlated with reduced steroid levels. Others have also measured low caloric intake in wrestlers (925-1,821 kcals/day) although total and free testosterone levels were not measured (Short and Short, 1983). Ayers et al. (1986) reported significantly reduced total testosterone in 14 out of 20 marathon runners and significantly reduced total and free testosterone levels in an "anorectic sub-group" concerned with lean body mass and caloric intake. Others have examined dietary intake in relation to total and free testosterone levels during non-exercise conditions.

Hill et al. (1980) reported significantly reduced total testosterone in men on a vegetarian diet and Hamalainen et al. (1983) reported significantly reduced total and free testosterone in normal healthy men on a low fat high fibre diet. Klibanski et al. (1981) observed a decrease in total testosterone in men after a 10 day fast. In another investigation a carbohydrate supplemented 1500 kcal/day diet in men resulted in a significant reduction in testosterone levels. Three regimens of carbohydrate refeeding did not elevate testosterone levels (Nan Hokyung et al., 1981). Zubiran and Gomez Mont (1954) reported low testosterone levels in Mexican males on a nutritionally inadequate diet. Anorectic men also exhibit pathological levels of total and free testosterone (Brown, 1983) which are normalized on refeeding (Beaumont 1972).

The precise mechanism by which the HPG axis of men and women runners adapts to exercise is not clear. However, recent concerns with the dietary intake of men and women in exercise programs raise questions with regard to the energy balance of habitual exercisers and more specifically runners (Rosen et al., 1986; Strauss et al., 1985; Brownell et al., 1988).

A consistent omission in investigations of the effects of exercise on the HPG axis in men and women in exercise programs or under chronic training conditions is the measurement of caloric intake and/or energy balance. However, there is considerable evidence to suggest that men and women undergoing programs of repetitive endurance training maintain high levels of activity on apparently calorically deficient dietary intake. Additionally alterations in dietary practices may contribute to alterations in the endocrine system in men and women.

Recent investigations of men (Short and Short, 1983) and women (Drinkwater et al., 1984; Marcus et al., 1984; Nelson et al., 1986) have suggested that caloric intake would appear to be inadequate in relation to activity and training levels.

Short and Short (1983) studied dietary intake over a 4 year period in 10 men's and 6 women's varsity teams. Although footballers demonstrated high caloric intake values (5,270 kcals/day) the dietary intake of male wrestlers was as low as 925 kcals per day during the competitive season. Drinkwater et al. (1984) reported daily caloric intake levels of 1,750 kcals/day in women training 33.4 miles/week and Marcus et al. (1985) reported dietary intake levels of 1,400 kcals/day in a group of women marathon runners training 65 miles per week. Nelson et al. (1986) reported caloric intake levels of 1,990 kcals/day in a group of 28 women runners training at least 37 miles/week. A three day diet diary recording technique was utilized in all the above investigations. Caloric intake would appear insufficient to support such activity levels. Caloric intake was relatively low compared to a non-athletic population (Liebell and Hirsch, 1984). The food efficiency factor (kcals/kg) was lower in the runners cited in the above investigations (31.9; 31.1; 35.4 kcals/kg/body weight respectively) than the sedentary controls v (36.4 kcals/kg/body weight) of the Liebell and Hirsch (1984) investigation. The following questions are therefore pertinent:

Does involvement in running lead to alterations in dietary behaviour in men and women?' and in addition do alterations in dietary practices contribute to exercise associated changes in the endocrine system?

It is clear that starvation contributes to pathologically low testosterone levels in male anorectics (Beaumont, 1972) and starving Mexican men (Zubiran and Gomez-Mont, 1954). It is also clear that starving anorectics are often hyperactive (Litt and Glader, 1986). The effects of the interaction of nutrition and training stress on the HPG axis in men and women have yet to be clarified.

The Effects of Exercise on Caloric Intake:

Human Investigations:

Correlational studies suggest that under exercise conditions humans decrease their caloric intake. Edholm et al. (1955) demonstrated that army recruits consumed less food on days of military training. The authors did, however, fail to consider the time available for food consumption. In a classic investigation of 213 millworkers in India, Mayer, Roy and Mitra (1956) found that food intake was greatest in the heavy manual labourers but that moderate intensity work was associated with lower caloric intake than in sedentary office workers. Factors such as meal time available were not considered as this may have related to opportunity to eat. Katch, Michael and Jones (1969) reported a moderate decline in caloric intake in 15 previously inactive college women taking part in either a daily tennis or swimming program. Caloric intake decreased from 1,751 kcal/day to 1,584 kcal/day in 20 college age women taking part in a 10 week exercise program and Watt, Wiley and Fletcher (1972) reported a decrease in caloric intake from 2,867 to 2,088 kcal/day after a 12 week exercise program in 30 middle aged men.

Conversely others have reported no changes in caloric intake with programs of exercise. Dempsey (1964) found a non-significant 100-200 kcal/day increase in a group of overweight men taking part in an 18 week exercise program. However, since an hour of activity may utilize 400-600 kcals of energy then a dietary deficit could be implied. Skinner (1964) also reported no changes in caloric intake in a group of men engaged in a 6 month calisthenics and exercise program. Again a caloric deficit could be implied from failure to compensate for increased activity with increased caloric intake.

Food Intake in Athletic Populations:

Several cross sectional investigations indicate that involvement in training programs leads to calorically insufficient diets when caloric intake is compared to the Recommended Nutrient Intake Tables for Canadians and when activity level is taken into account (Short and Short, 1983; Drinkwater et al., 1984; Marcus et al., 1985; Nelson et al., 1986). Although such investigations cannot imply cause due to their correlational nature, they do suggest that training is associated with low caloric intake.

Others have suggested that the adoption of a training program does not result in an increase in caloric intake to compensate for increased activity (Wood et al., 1983).

Short and Short (1983) reported caloric intake as low as 925 kcals/day in wrestlers and Marcus et al., (1985) reported caloric intake levels of 1,440 kcals/day in a group of women marathon runners training at least 65 miles/week. Others have reported slightly higher levels of caloric intake in women runners (Drinkwater et al., 1984; 1,750 kcals/day and Nelson et al., 1986; 1,990 kcals/day) although intake would still be considered below the RNI levels for Canadians.

In contrast some authors have reported higher levels of caloric intake in men and women runners (Blair et al., 1981). Blair et al. (1981) reported that men and women runners consumed 2,959 and 2,386 kcals/day respectively.

Animal Investigations:

Animal investigations have suggested that free or forced exercise under free feeding conditions is associated with a decrease in caloric intake (Mayer et al., 1954; Oscai, Mole and Holloszy, 1971; Stevenson et al., 1966; Levitsky, 1974; Oscai and Holloszy, 1969; Ahrens et al., 1972; Crews et al., 1969) However, this decrease in food intake may be temporary since it has been demonstrated that after the initial 5 or 6 days of running, food intake recovers to above baseline levels (Tokyama et al., 1982). Decreased appetite in exercise animals appears to be associated with exercise intensity (Oscai, 1973; Katch, Martin and Martin, 1979).

Mayer et al. (1954) subjected normal rats and genetically obese mice to 20,40 and 60 minutes of swimming. Exercise was followed by a slight decrease in caloric intake which was followed by an appetite increase. Oscai,Mole and Holloszy (1971) reported that forcing rats to swim resulted in a decrease in food consumption in female rats compared to control animals. There was no effect of exercise on male animals. Stevenson et al. (1966) forced rats to run which resulted in decreased caloric intake. This behaviour was maintained when exercise was irregular and not presented as a fixed interval food schedule. A finding of decreased caloric intake in animals under exercise conditions has also been confirmed by others (Oscai and Holloszy, 1969; Crews et al., 1969; Ahrens et al., 1972). It would appear that the *opportunity to exercise* alone versus forced exercise will result in reductions in food consumption. However, the non-specific effects of exercise induced stress cannot be ruled out in appetite suppression with exercise. Premack and Schaeffer (1963) introduced a running wheel to free feeding rats and produced a reduction in food intake in the first 7 days following the introduction of the running wheel. This was confirmed by Levitsky (1973). Epling and Pierce (1984) demonstrated that opportunity to run and daily rate of change of activity levels were associated with reduction in food consumption in animals. Animals were permitted varied access to wheel running and subjected to a 90 minute food schedule. Strong anorexia (food intake reduction) occurred when opportunity to run exceeded 12 hours. Food intake therefore declined as a function of opportunity to run.

The decrease in food intake associated with the introduction of a running wheel may be temporary since it has been demonstrated that after the initial 5 or 6 days of running food intake recovers to above baseline levels (Tokyama et al., 1982; Mayer et al., 1954).

Exercise *intensity* appears to affect caloric intake in animals under forced or free exercise conditions. Oscai (1973) reported that food intake suppression was proportional to exercise intensity in male animals. Katch et al., (1979) observed that short duration high intensity exercise reduced caloric intake to a greater extent than extended duration low intensity exercise. Human investigations have supported a finding of an acute high intensity exercise mediated suppression of appetite. Thompson et al. (1988) examined the effects of 12 hours of fasting on caloric intake (20 minute) and sucrose palatibility and appetite in 15 college age men. Subjects underwent a control non-exercise, and two exercise periods (35% and 65% VO2 max.). The high intensity exercise period was associated with a short term reduction in appetite.

Others have suggested that *activity affects the duration of feeding* rather than food consumption per se (Kanarek and Collier, 1979; 1983) and Pierce et al. (1986) provided evidence that the desire for feeding is reduced by activity. Prior bouts of exercise either voluntary or forced reduced the reinforcing effectiveness of food for bar pressing in animals.

In summary it appears that free or forced exercise and daily rate of change of activity are associated with reductions in food intake. Reductions in food intake may be due to satiety (stopping eating once started) or to a reduction in the reinforcing effectiveness of food.

The Effects of Food Intake on Activity Levels in Humans and Animals:

The Effects of Food Intake on Activity in Humans:

It appears that food availability may affect activity levels in humans and animals (Epling and Pierce, 1988). Increased mobility has been associated with human starvation (Howard, 1839; Russel Davis, 1951). Mobility may be due to physiological changes that are in part a result of evolutionary processes. Mobility in times of starvation has survival value since to remain in areas of inadequate food supply could be detrimental to the health of the organism.

The Effects of Food Intake on Activity in Animals:

Experimental evidence suggests that food deprivation affects levels of wheel running in animals (Epling and Pierce, 1988). Depriving rats of food increases the level of wheel running when animals are given the opportunity to exercise (Cornish and Mrosovsky, 1965; Finger, 1951; Hall and Hanford, 1954; Reid and Finger, 1955). It would appear that food restriction alone is sufficient to increase physical activity in animals (Epling and Pierce, 1988). Food schedule may also affect activity levels in animals.

The Effects of Food Schedule on Activity Levels in Animals:

When rats and mice are fed once per day for 60 - 90 minutes they initially lose weight but over a few days adjust to the new level of food availability (Epling and Pierce, 1988).

Animals exposed to similar food schedules but allowed free access to running wheels except while being fed continue to lose body weight, increase their running and die (Hall and Hanford, 1954; Spear and Hill, 1962; Routtenberg and Kuznesof, 1967; Routtenberg, 1968; Epling et al., 1981). Routtenberg and Kuzneszof (1967) demonstrated that rats allowed free access to a running wheel and a 60 minute feeding schedule increased their wheel running, self-starved and died. Chloropromazine (CPZ) (an appetite stimulant and hypoactive agent) stimulated appetite while at the same time decreasing activity. Survival rate under CPZ administration increased to 75%. Routtenberg (1968) habituated rats to the running wheel prior to food restriction. This increased the survival rate among animals on the 60 minute food schedule with opportunity to exercise although it did not eliminate the anorectic effect of food schedule and exercise. Epling et al., (1981) extended this effect to mice. Excessive wheel running in rats has been noted when the number of meals was changed from free feeding to one meal per day (Routtenberg, 1968; Spear and Hill, 1962; Routtenberg and Kuzneszof, 1967).

A possible criticism of such studies is the accidental reinforcement of running by food scheduling (Epling, Pierce and Stefan, 1983). However, Epling et al., (1983) observed that in the investigation of Kuzneszof (1967) starvation occurred even when access to running was delayed following feeding.

Thus Epling and Pierce (1988) suggest that: 1) activity appears to be associated with decreased caloric intake and 2) that increased activity appears to be associated with food scheduling and/or food deprivation.

A Theory of Activity Anorexia: An Evolutionary and Physiological Perspective

Recently Epling and Pierce (1988) have proposed a model of Activity Anorexia based on a biophysical perspective. This model, which they argue may generalize to humans, is based on:

1) A decrease in caloric intake with activity.

2) An increase in activity with food scheduling and/or deprivation.

3) Daily rate of increase in activity.

It is suggested that humans, particularly those involved in the modern "exercise culture" are at risk for anorexia. Additionally the culture provides necessary and sufficient conditions for activity anorexia (Epling et al., 1983; Epling and Pierce, 1988). The activity anorexia model assumes that exercise may be initially motivated by a desire to lose weight or gain fitness and may be summarized thus:

1) Increased physical activity decreases food intake or decreased food intake increases activity.

2) The decreased value of food reinforcement results in a decline in both food intake and body weight.

3) As body weight decreases the motivational value of exercise increases (as weight is lost incentive to increase exercise further is enhanced).

4) A further escalation in activity results in a further decrease in the reinforcement value of eating.

5) A cycle of increased activity and decreased caloric intake is established which is extremely resistant to change.

A model which offers an explanation of dietary disorders commonly associated with habitual exercise must explain the tendency to exercise under conditions of food restriction and the interrelationship between exercise and eating. Furthermore, the model must be shown to be generalizable to humans. Epling and Pierce (1988) have produced a model of Activity Anorexia based on an evolutionary and physiological perspective.

Activity Anorexia: An Evolutionary Perspective:

The evolutionary analysis of activity anorexia is based on the fact that man and other animals become mobile in times of food scarcity. This had survival value since remaining in areas where food is scarce would result in death by starvation. Once food is located then it would be feasible to assume that both man and animals would then decrease activity *unless a further stimulus to maintain activity was present.* Animals that became active thus survived and this genetic compliment was passed on to further generations. Arguably cultural reinforcement of low caloric intake and excessive activity patterns could induce activity anorexia.

Activity Anorexia: A Physiological Perspective:

A physiological perspective is based on the role of the endogenous opiate peptides, specifically beta-endorphin (BEP). Since food deprivation *increases* the reinforcing effectiveness of running and running *decreases* the reinforcing effectiveness of food this suggests a physiological mechanism linking the motivational value of eating and running (Epling and Pierce, 1988).

Endogenous and exogenous opiate stimulation appears to increase appetite under conditions when body weight is normal. When weight is low (ie: declining during exercise programs or during starvation) BEPs appear to function as appetite suppressants or alternatively some other mechanism antagonizes the appetite stimulating effects of the BEP system.

The injection of BEP into the hypothalamii of non-hungry animals stimulated feeding (Grandison and Guidotti, 1977; Liebowitz and Hor, 1982; McKay et al., 1981). The effects of BEP on feeding in animals has generally been examined indirectly via the administration of the opiate antagonists, naloxone (NLX) and naltrexone (NTX) which attenuates feeding and drinking behaviour in animals (Fishman et al., 1983; Holtzman, 1975; 1979; Kirkman and Blundell, 1984; Maickel et al., 1977; Margules et al., 1978). The effects of the BEP system on feeding may in part be mediated by the dopaminergic system (DA) since DA inhibits dynorphin induced feeding in animals (Morley and Levine, 1983; Morley et al. 1982).

The interaction of opiates, exercise and feeding has been examined by Davis et al. (1985a; 1985b) under acute and chronic exercise conditions although the evidence for an opiate mediated effect on appetite under exercise conditions is unclear. Evidence from the Davis et al. investigations has suggested:

1) A short term (0-2 hour) opiate mediated hyperphagia which is NTX reversible.

2) A period 2 (2 hours +) opiate deficiency mediated hypophagia which is not altered by NTX.

3) A medium term (4 week) opiate deficiency mediated hypophagia associated with a reduction in 2 deoxy-D-glucose (opiate dependent) induced feeding.

4) Long term (12 week) opiate mediated appetite control since BEP levels were elevated at 12 weeks of training and NTX attenuated food intake in a dose dependent manner.

Others have reported that opiates have a suppressive effect on eating . Sanger and McCarthy (1980) observed that food deprived animals (ie: low weight) ate less food when injected with morphine. This suggests that opiates may act to decrease appetite under food deprivation conditions (Epling and Pierce, 1988). Alternatively the opiate appetite enhancing effect may be antagonised by other substances released under starvation conditions when body weight is low.

Epling and Pierce (1988) also interpreted the Davis et al. (1985 a&b) as producing evidence of an opiate suppression of appetite under exercise conditions since injections of 2 deoxy-D-glucose (opiate stimulus) resulted in lower food intake in trained versus untrained animals after a training period.

An opiate mediated suppression of food intake associated with low body weight is supported by evidence from patients with anorexia nervosa. Elevated cerebro-spinal fluid levels of BEP have been reported in anorectics (Kaye et al., 1982) and NTX stimulated weight gain in anorectic patients (Moore, 1981). This effect may be reinforced by excessive activity which is often observed in anorectic patients (King, 1963; Kron, 1978; Crisp, 1965; Crisp, 1980; Litt and Glader, 1986). Periods of activity are associated with elevations of plasma BEP in non-clinical populations (Appenzeller, 1980; 1981; Farrell, 1981;1985; Farrell et al., 1981;1982). Since activity may precede the onset of anorexia nervosa and abnormal psychopathology (Katz, 1986) then this would support a model of opiate mediated activity anorexia in normal individuals.

Appenzeller (1980) reported a surge in in BEP lasting 2 hours following a marathon and Farrel (1981) reported an increase in BEP at an exercise intensity of 60% of VO2 max. Elias et al. (1986) examined BEP and Beta-Lipotropin (BLPH) secretion before and after 20 minutes of sub-maximal exercise at 80% VO2 max. in 8 men. An exercise associated increase in BEP/BLPH returned to normal 60 minutes post exercise. Colt (1981) reported an increase in BEP proportional to degree of effort in 26 long distance runners after a hard bout of running and Elliot et al. (1984) reported a significant increase in BEP/BLPH in 5 men following a bout of treadmill running and weight lifting.

Research thus suggests:

1) An opiate mediated decrease in food intake under starvation conditions when body weight is low.

2) At normal body weight and under normal free feeding conditions opiates act to increase appetite.

It should however, be considered that measurement of opiates in peripheral circulation may not necessarily reflect opiate mediated appetite effects occurring centrally (specifically in the hypothalamic area) and separated by the brain-blood barrier.

To date there has been no investigation of the interaction of training programs, weight loss, and opiate mediated effects on appetite in relation to supporting a model of exercise associated anorexia in men nor women.

Alterations in Metabolic Rate and Food Efficiency With Food Deprivation and Training: Relevance to a model of human Activity Anorexia.

It is well established that anorectic patients maintain high levels of activity on very limited caloric intake. The basal metabolic rate (BMR) is depressed in the anorectic patient and the efficiency of nutrient metabolism enhanced. This mechanism serves to protect the anorectic (albeit temporarily) from the effects of increased energy expenditure and decreased caloric intake. If a model of activity anorexia in humans is tenable then evidence must be presented to suggest adaptive metabolic alterations to increasing exercise and food deprivation.

Non-exercise based investigations with normal and obese individuals suggest that caloric deprivation decreases BMR (Bray, 1969; Welle et al., 1984; Lammert and Hansen, 1982). A 15% reduction in BMR was reported by Bray (1969) in obese women on a 450 kcal/day liquid diet and a 9.5% reduction in BMR was reported by Welle et al. (1984) in women on a 472 kcal/day diet. Lammert and Hansen (1982) examined the response of over and underfeeding and normal caloric intake on BMR. Semi-starvation was associated with a decrease in BMR compared to other feeding conditions.

To date there is little agreement on the effects of training on the BMR of athletes (Brehm, 1988; Brownell et al., 1987). Stern et al. (1980) reported that exercise attenuated the suppression of BMR induced by caloric deprivation of

500 kcal/day. In contrast Phinney (1985) reported that dietary restriction decreased BMR by 10% and that a combination of diet and exercise decreased BMR by 30%. Warren (1988) investigated the role of energy balance in the development of amenorrhea. Amenorrheic runners were reported as having a lower resting BMR than normally cycling runners. Warren concluded that high mileage runners did not increase caloric intake to compensate for the increased energy expenditure of activity but maintained energy balance through a reduction in the BMR.

In times of dietary restriction food efficiency in animals (Brownell et al., 1987) and humans (Leibel and Hirsch, 1984) is elevated. Food efficiency may be defined as the ratio of food ingested to body weight and represents an index of nutrient requirements for the maintenance of body weight (Brownell et al., 1987). By definition food efficiency increases when calories required to maintain body weight decreases. Under dietary restriction it would therefore appear that the human organism is able to gain more energy from food than under normal feeding circumstances (presumably by enhanced digestive efficiency and decreased gastric motility).

This is of particular relevance to a model of activity anorexia for two reasons:

1) The activity anorexia model assumes that weight loss provides a motivation for increasing exercise. Subsequently weight loss and possible opiate mediated appetite suppression effects decrease the reinforcing value of food. Since food efficiency increases as food intake decreases then weight loss would become more difficult. This would add to motivation to increase exercise and is consistent with the case histories described by Katz (1987).

2) Increased efficiency of nutrient utilization would permit the development of activity anorexia with pathological weight loss and the maintenance of

extreme activity levels under conditions of dietary deprivation without apparent harm to health for an extended period..

In summary, a decrease in BMR and an increase in food efficiency with exercise and dietary restriction are consistent with a model of activity anorexia in humans and would account in part for the stable and resistant cycle described by Epling and Pierce (1988).

The Endogenous Opiate Peptides and the Effects of Exercise on the Hypothalamic-Pituitary-Gonadal Axis in Men and Women.

Chronic involvement in running training programs is associated with alterations in the HPG axis in men (Wheeler et al., 1984) and women (Cumming, 1987). Nutritional factors may play a role in these alterations (Cumming, 1985) and excessive running in women has been associated with anorectic eating attitudes (Henry, 1982). Exercise programs are effective in reducing body weight and body fat (Depres et al. 1980) and exercise programs may result in decreased appetite. Exercise increases circulating BEP levels and this has been associated with decreased appetite under conditions of low body weight such as in anorexia nervosa (Epling and Pierce, 1988) although it would not appear that BEPs are associated with the endocrine changes accompanying endurance training (Cumming and Wheeler, 1987). Such alterations in the HPG axis in runners are consistent with changes observed in anorectic individuals (Brown, 1983) and starving men (Zubiran and Gomez-Mont, 1954), although similarities are qualitative rather than quantitative. Pathologically altered testosterone levels were observed in an "anorectic sub-group" of male runners consistent with levels in anorectic male patients have been observed (Ayers et al.,1986). It is possible that an interaction of activity with the BEP system and alterations in appetite and/or food intake may be responsible for alterations in the HPG axis of runners. However, since peripheral measures of BEP levels may not be indicative of central levels then any association of BEP's with appetite is tentative.

Most investigations of the effects of the BEP system on endocrine function in men and women have not considered an interaction of exercise and caloric intake on the HPG axis.

Investigations of the effects of the BEP system on the HPG axis in men and women have utilized the opiate antagonists NLX and NTX or morphine. Studies in women have suggested an inhibitory role of the BEP on the HPG axis. Morley et al. (1980) observed that NLX given to normal women increased LH levels and Ropert Quigley and Yen, (1981) demonstrated that administration of NLX for 6 hours increased LH pulse frequency and amplitude. Quigley, Sheehan, Casper and Yen (1980) examined the interaction of the DA and BEP systems in 4 amenorrheic and 4 normal cycling women. NLX and MCP administration had no effect on LH patterns in normal women although serum LH was increased in 4 women with amenorrhea and decreased in 4 amenorrheic subjects. Delitala, Devilla and Musso (1983) demonstrated an NLX induced increase in LH in women which was suppressed by DA. Similar findings have been reported in men. Morley et al. (1980) reported an NLX induced LH rise in men and Veldhuis et al. (1984) reported that even with a suppressive dose of steroids NLX reinstated LH pulse frequency in men. Bergjelk et al. (1986) observed that an NLX infusion resulted in an increase in serum LH in normals and transexuals which was not altered by the administration of suppressive doses of estrogens. Elias et al. (1986) demonstrated that a GnRH stimulated LH increase in men was augmented by an NLX infusion. Others have reported no effects of NLX on LH secretion in men

(DeFeo et al., 1986; Martin et al., 1985). Pfeiffer et al., (1986) reported no effect of Kappa receptor agonists on LH secretion in normal men. Contrasting findings may in part be related to dose specific responses of the HPG axis to NLX induced opiate receptor suppression in men (Knuth and Neisschlag, 1985; Martin et al., 1985). Although NLX stimulated LH secretion and DA suppressed the NLX induced rise in LH, pre-treatment with metaclopramide (MCP) failed to alter the magnitude of LH increments observed during NLX infusion. This did not suggest a role for DA receptors in NLX induced LH changes in men (Delitala, Devilla and Musso, 1983).

Evidence of an opiate mediated effect on the HPG axis has not been substantiated under exercise conditions. Rogol et al. (1984) demonstrated an NLX mediated increase in LH secretion in runners and non-runners and suggested that there were no differences in opiate mediated control of the HPG axis between the groups. Elias et al. (1986) reported that exercise failed to modify the LH/FSH response to GnRH and NLX failed to alter post exercise or GNRH stimulated LH levels. Although decreased LH pulse frequency (2.2 ± 0.48 v 3.6 ± 0.24/8 hours), amplitude and LH response to GnRH was reported in male runners versus sedentary controls, the role of the BEP system was not investigated (MacConnie et al., 1986).

Although the bio-physical model offers an attractive explanation of the Activity Anorexia model as it may generalize to man, there remains only correlatory evidence of an evolutionary and physiological explanation of the interraction of appetite and exercise.

Other Factors in Support of an Activity Based Anorexia in Humans:

Epling and Pierce (1988) suggest that certain conditions are necessary and sufficient for the development of an activity anorexia in humans:

1) A decrease in food intake with exercise.
2) Opportunity to exercise.
3) A change in attitude towards food (decreased reinforcement value).
4) Decline in body weight.
5) Evidence of the development of increased motivational value of exercise.
6) A process of exercise and dieting which is resistant to change.

Furthermore there are:

7) Recent reports of exercise induced anorexia.

Decreased food intake with exercise and alterations in attitudes towards food:

Although various investigations have suggested a decrease in caloric intake with training programs in men and women (see previous discussion), there has been a general failure to report changes in attitudes towards eating. Recently obligatory or habitual runners have been compared to the anorectic patient in terms of psychological profile and attitudes towards body fat and food intake (Yates et al., 1983; Sours, 1981) although this *disease model* has been refuted in favour of a model of *affect regulation* (Blumenthal et al., 1985). Others have described anorectic eating attitudes (Henry, 1982) and pathological weight control behaviours (Rosen et al. 1986) in women runners. A food aversion simulating anorexia nerovsa has been described in high school age athletes (Smith, 1982) and an anorectic sub-group has been described in an investigation of the HPG axis in male runners (Ayers et al. 1985). Others have attributed severe weight loss and pathological levels of total and free serum testosterone to inadequate caloric intake in wrestlers (Strauss et al., 1985). Others did not find anorectic eating attitude scores in men (Wheeler et al., 1984) and women (Weight and Noakes, 1987) runners. It is unrealistic and erroneous to compare the severe psychopathology of anorexia nervosa with the apparently healthy aspects of regular running. However, there would appear to be considerable concern with food and body weight among runners. For the want of an adequate model, early accounts have compared the group with the anorectic. There is little doubt that the runner shares a similar concern with exercise as do many anorectics but until the model of Epling and Pierce (1988) there has been no adequate model to explain the relationship of food intake with exercise and exercise with food intake. Of great significance here is the relationship to the evolutionary perspective of running. The reader will recall that activity increases during times of food scarcity in animals and man. Activity, should in theory, decrease when food is located. It is feasible that the cultural reinforcement associated with self-control with regard to eating may serve to limit caloric intake even when food is available. Epling and Pierce (1988) observe that cultural pursuit of the perfect body image may contribute to internalization of societal values and standards and anorexias. As weight decreases running becomes easier and therefore potential for increasing activity occurs.

Exponential Increases in Running, Motivational Value of Exercise and the Development of a Condition Resistant to Change:

Running is an effective means of weight control. Running programs have proved effective in lowering body weight and decreasing percent body fat. (Depres et al., 1985).

As body weight falls then arguably the reinforcing effectiveness of food intake would decrease. This would be enhanced by an apparent appetite suppression effect of exercise. Conversely the reinforcing value of exercise increases since weight loss is achieved. Since food efficiency has been shown to increase and BMR has been shown to decrease under conditions of dietary restriction and exercise then weight loss would be slowed. This may provide further motivation for an increase in activity. The significant increase in the incidence of overuse injuries in runners (Stanish, 1984; Clements et al., 1981; Hikida et al., 1983; Norregard-Hansen et al., 1981), the inability of the runner to cease running even when given medical advice associated with negative addiction (Morgan, 1979) and neurotic illness in fitness fanatics (Little, 1981) support the development of a condition which is extremely resistant to change. If the runner is also preoccupied with caloric intake then an important association with the Epling and Pierce (1988) model would seem apparent.

Reports of Development of Anorexia following the Onset of an Exercise Regimen:

Some of the most convincing recent documented evidence to date of the development of an Activity Anorexia in humans is provided in two case reports by Katz (1986). Katz (1986) documents two cases which may briefly be summed up in the following:

1) Distance running was adopted as a means of getting in shape by a physician and ex-athlete.

2) No previous history of eating disorders or psychopathology was apparent in either subject.

3) There was a rapid increase in training distance accompanied by a dramatic weight loss in both subjects.

4) Running distance continued to increase and weight continued to fall accompanied by an increasing concern for body weight and the caloric content of meals.

5) In case one, a forced reduction in running due to vocational commitment led to the development of a depressive condition during which the subject became further preoccupied with weight loss and caloric intake. This was accompanied by the onset of bulimic episodes which increased in frequency.

6) Case two's running performance began to decrease even with increased training and the eventual development of a knee injury curtailed running. A state of depression ensued and bulimic behaviour developed simultaneously with this depressed state. Further depression occurred as concern for body weight grew.

7) Both developed a psychopathology so severe that medical advice was sought and the individuals presented to a physician.

8) From the onset of exercise to the time when the individuals sought medical advice, case one lost approximately 35% of initial body weight and case two 17% of initial body weight. It should be borne in mind that in the case of the subject two, initial body weight was low since the subject was a wrestler.

These case histories provide evidence of the development of an anorexia and depressive psychopathology associated with exercise. This has also been substantiated by Keys et al. (1950) who demonstrated the development of neurotic traits and increased activity in a group of concientious objectors subjected to an extended period of starvation.

The current societal preoccupation with exercise, dieting and slimness would therefore appear to provide the necessary and sufficient conditions for the development of an Activity Anorexia syndrome. Arguably conditions diagnosed as Anorexia Nervosa today may infact be activity anorexias. The fundamental difference is that the medical profession attributes the activity of anorexia nervosa as a means of weight control whereas a hypothesis of activity anorexia in humans is based on activity as a fundamental etiological factor in the development of a condition which is highly resistant to change. Many anorexia nervosa cases may be anorexias which are a function of exercise and not a premorbid personality disorder.

Purpose of the Investigation:

The purpose of the following investigation was:

1) To investigate the effects of a six month training program on indices of fitness and anthropometric measures on healthy men and women and experienced high mileage runners. The exercise program was implemented to;
2) To examine one aspect of the activity anorexia model according to Epling and Pierce (1988); that is the effect of exercise (six month running program), opportunity to exercise and unlimited food intake to run on caloric intake.
3) To investigate the role of exercise and nutrition in changes in the HPG axis in men as this supports a model of human caloric restriction and/or negative energy balance.

To examine these questions a six month running program was designed to improve the training volume of previously sedentary healthy adults to 25 - 30 miles/week (40 - 56 km/per week). Measures of weight, body fat and maximal aerobic capacity were performed as a validation criterion for the training program.

Specific Objectives of the Proposed Research.

The primary purpose of research is to examine theory; to provide support or evidence against a particular hypothesis or hypotheses. In accord with this view the following research project has attempted to contribute to a theory of human activity anorexia in men and women. Inherent in this objective was the identification of conditions which may result in men and women changing their dietary patterns as a consequence of opportunity to engage in a progressive exercise program. The following were the specific experimental objectives of the proposed project:

1) To investigate the effects of high mileage running training on nutritional intake in men and women and hypothalamic-pituitary-gonadal axis in healthy adult men.

2) To investigate the effects of a six month training program on the dietary intake of healthy sedentary men and women.

3) To investigate the effects of a six month training program on the hypothalamic-pituitary-gonadal axis in healthy adult men.

4) To investigate the relationships between nutritional and hormonal variables among healthy men and women involved in a training program and chronic exercise routines.

5) To examine the effects of a standardised exercise program on dietary intake in healthy sedentary men and women as it relates to a model of Activity Anorexia; that is; free food availability and opportunity to exercise results in appetite suppression.

Hypotheses:

Five main hypotheses were tested:

1) Six months of endurance running training will result in significant reductions in caloric intake in previously sedentary healthy men and women. High mileage men and women runners will consume significantly less calories than non-exercisers despite their activity level.

2) Six months of endurance training designed to increase the weekly training load of a group of healthy sedentary men and women to a mean of 40 to 56 km per week will result in a decrease in circulating testosterone in the men. Furthermore, high mileage runners will demonstrate significantly lower total testosterone levels than the sedentary male subjects.

3) Six months of endurance training will result in alterations in the pulsatile release of LH in the male training group. Alterations will include decreased LH pulse frequency, decreased pulse amplitude and area under the LH curve. Furthermore, LH pulsatile characteristics will be significantly different between high mileage runners and sedentary but healthy men.

4) Alterations in testosterone will be related to alterations in LH pulsatile release in the training group.

5) Alterations in total testosterone and LH characteristics will be related to caloric and macro-nutrient intake and changes in caloric intake.

Chapter 2.

Methods and Procedures.

Subjects:

Three groups of subjects were invited to take part in the study. Group one comprised 10 high mileage male runners (HMR) and 10 high mileage women runners (FHMR) training at least 48 kilometres per week. Group two were 20 men and 20 women invited to take part in a six month progressive jogging programme (TRM and TRF respectively) designed to increase their weekly mileage from zero to approximately 40-56 kilometres per week. Group three was a control group comprising 15 men and 15 women who had no interest in, and who took no part in any organized exercise programmes. Participants in the study were chosen from respondents to three separate advertisements. The first advertisement called for high mileage runners training at least 48 kilometres per week to take part in a study to monitor their fitness level, endocrine profile and dietary habits during a six month period. The second advertisement solicited healthy men and women to take part in a six month jogging programme including the monitoring of nutritional behaviour and endocrine variables. The third advertisement was directed towards healthy men and women to take part in a study of the dietary behaviours of sedentary men and women. In this third advertisement it was stressed that involvement in the study depended on a complete lack of interest in, and participation in any organized exercise programme. A financial incentive package for control subjects was outlined. All advertisements were run in the student newspaper, the Gateway, and the University of Alberta Folio magazine. Subjects were selected from respondents to the advertisements according to the following criteria:

Controls:

Respondents to the advertisements were interviewed individually following an initial phone interview regarding exercise habits. An activity questionnaire was completed to ensure that individuals were suitable for the study. Thirty individuals were selected, 15 men and 15 women, and were invited to attend a final selection/screening meeting to ensure that participation criteria were met. All inconsistencies were checked and any doubt as to the credibility or reliability of the individual resulted in elimination from the subject pool and the selection of another subject. Those selected were then invited for two orientation meetings for completion of questionnaires, physical fitness testing, and instruction in the completion of 3 day diet diaries. A form of informed consent was completed and the obligations of the participants to the investigation fully outlined. Following all the screening procedures and orientation meetings the investigator was satisfied that credible controls had been selected from the potential subject pool. It is recognized that a true control group is randomly sampled from the population at large. This ensures a homogeneity of variance among extraneous variables which may affect the experiment ie. error. However, the nature of the following investigation necessitated the selection of controls from a respondent pool, since it was vital to the investigation that the subjects chosen were truly sedentary. Since funds were available for an incentive it was found that several individuals attempted to infiltrate the investigation for the purpose of financial gain although they were in fact well trained individuals.

Such potential participants were not retained within the experimental subject pool. For the purposes of the script this group will be called *the control group* although violations of the true term are recognized by the author.

High Mileage Runners:

Respondents to the advertisement for high mileage runners were interviewed and selected according to their fulfilling the following criteria:

(a) Training year round and averaging approximately 48-56 kilometres per week.

(b) Having been training over these distances for at least two years.

(c) Able to complete the investigation.

Training Groups:

All those responding to the advertisement were interviewed on the phone and promptly made aware of the degree of commitment expected during the course of the study. The activity levels of the respondents were investigated during the same phone interview to ensure that training state was negligible. Those who were found to exercise more than once per week or who were involved in any regular programme of exercise were omitted from the subject pool. Any individual who expressed doubt at being able to commit the necessary time to the investigation was also eliminated from the subject pool. Potential candidates for inclusion in the study were invited to the University of Alberta Fitness Unit to take a short standardized test of physical fitness. All those attending were asked to complete an activity questionnaire and complete the Physical Activity readiness questionnaire (PAR Q). The test battery is described in the measures section. Any individual who was training once per week was informed that in future all training must be confined to running. Any potential candidate for the investigation who expressed an unwillingness to comply with this directive was asked to withdraw from the study.

Programme Incentives:

In order to maximize participant adherence, incentives were offered to all groups. High mileage runners were offered the incentive of free fitness tests and anthropometric measurement. In return the high mileage group was asked to continue training according to their own schedule and to record their dietary intake in a diet diary over a three day period during alternate weeks. Control subjects were offered 10 dollars per completed diet diary in return for accurate diary completion and the maintenance of a sedentary lifestyle. Controls were also informed that free fitness programming would be offered at the end of the study.

The training groups provided their own financial incentive for remaining in the study by depositing 100 dollars which they were informed would be returned with interest at the end of the study. However, trainees were informed that failure to complete the study for reasons other than injury or mitigating circumstances ie. family demise or job opportunity; would result in the loss of the deposit on a pro-rated basis. In the event of injury the full one hundred dollars was refunded

Group Orientation Meetings:

All those chosen for the study were asked to attend two orientation meetings: The first was for the purpose of informing participants of the requirements and timelines of the study, to complete the Eating Attitudes Test (EAT), the Eating Disorder Inventory (EDI) and to collect a baseline blood sample from the men. The second meeting was conducted for the purpose of instruction in the completion of diet diaries. Orientation meetings were held between the hours of 1600-1800.

During the first meeting the subjects were informed that this was merely a study of the effects of exercise on fitness level. No information was given with regard to appetite changes associated with exercise since we did not wish to influence the dietary behaviour of the subjects. Subjects were simply asked to record dietary intake during the course of the study. It was stressed that the utmost integrity should be followed in the completion of the diet diaries since failure to comply with this requirement would render the study inaccurate. Also during this meeting consent forms were signed and a document outlining all the potential positive and negative effects of exercise programmes was given to all subjects. No emphasis was put on any particular positive or negative effect of running. Any subject over the age of 35 was asked to provide a note from a physician to say they were medically sound and able to take part in an endurance running program. Any subject over the age of 35 was also required to undergo a cardiac stress test at the University of Alberta Hospital. Subsequent testing of such subjects was completed under the supervision of a physician. Written proof of the completion of a cardiac stress test was required prior to admission to the investigation.

A basal blood sample was taken from all the males in the HMR and TR groups between 1600-1800 hours and at least three hours after a light caffeine free lunch. All blood samples were taken at least 24 hours after any exertion. All subjects from all groups completed the EAT and The EDI.

At the second meeting a nutritionist conducted an instructional session on the completion of 3 day diet diaries. Such topics as meal size, estimation of portions and recording techniques were covered. Particular attention was paid to the minimization of error in the recording of dietary information and the importance of carrying the diary around during the day. Each subject was informed that dietary information was to be recorded after each meal. Subjects were also informed that the investigator would spend time with each of them to ensure accurate recording of dietary intake.

A Summary of Measurements:

Training Study Participants:

1) Eight minute sub-maximal test of cardiovascular endurance (Modified Astrand Protocol, U of A Fitness Unit). This test was used to estimate maximal aerobic power and was used in place of a direct assessment for safety reasons.
2) Skinfold measurements were taken to give an estimate of degree of adiposity and to establish a percentile rating for each subject against national norms.
3) Height and weight were measured.
4) Subject selection was based on results (overweight, high blood pressure).

Pre, Mid and Post Programme Measurements: All groups:

1) Maximum Aerobic Capacity: Direct measurement via treadmill protocol and the Beckman MMC according to protocols outlined in Physiological Testing of the Elite Athlete, MacDougall, Wenger and Green, 1982).

2) Percentage Body Fat: Estimated by the body density method (densitometry) utilizing the equation derived by Brozek et al. (1963), and assuming the two compartment model (fat and non-fat compartments).

Pre and Post Programme Measures: Controls:

Measures of eating behaviours and attitudes: (EAT and The EDI).

Pre and Post Programme Measures: Trainees:

1) Basal hormone levels (Testosterone, FSH, LH, Cortisol, Prolactin, SHBG).

2) LH Pulse Patterns (6 men as a sub-set from the training group).[1]

3) Measures of eating behaviours and eating attitudes: (EAT and EDI).

Pre and Post Programme Measures: High Mileage runners:

1) Measures of eating behaviour and attitudes: (EAT and EDI).

2) Baseline hormone levels.

---

[1] *LH pulse patterns will not be measured in the female training group since this data has already been provided by other investigators (Cumming et al. 1985).*

3) LH pulse patterns in a sub-set of 6 male volunteers.

Cross Sectional Investigations: All groups.

Pre and post cross sectional investigations of basal hormone levels were conducted on male high mileage runners and trainees.

Dietary Measurements: All groups:

All participants recorded their dietary intake over a one month baseline period. Two diaries were completed for three consecutive days (Thursday, Friday, Saturday) on alternate weeks. This established a basal level of caloric intake. Having established a baseline caloric intake all subjects were interviewed by the nutritionist to establish correct procedures in filling out the diaries and to correct any problems. Each subject was then instructed to complete diet diaries every alternate week for the designated three day period for a period of 24 weeks. All diaries were collected on a bi-weekly basis and were continually monitored by the nutritionist. Any problems arising were dealt with by personal or phone interview. Analysis of the diaries was completed by the author and a qualified nutritionist and expert in the use of the University of Alberta/Kellogg Diet Data Base (Kellogg-Salada Inc. 1980) comprising in excess of 20,000 name brand food items. The three day diet diary procedure has been used successfully in other investigations of caloric intake in athletic populations (Short and Short, 1983; Drinkwater et al., 1984; Marcus et al., 1985; Nelson et al., 1986).

Details of Measurement Procedures:

Physiological Measures:

Maximum oxygen uptake was measured directly in all subjects at three monthly intervals ie: pre, mid and post programme. The Beckman Metabolic Cart (MMC) and a standardized computerized metabolic programme was used: A modified version of a standard treadmill protocol was used for the determination of maximum oxygen uptake (MacDougall, Wenger and Green, 1982).

Prior to testing all trainees and control subjects were familiarized with the treadmill. Any of the high mileage group who were unfamiliar with the treadmill were also required to familiarize themselves with the machine. Orientation procedures were conducted in the following manner:

1) Subjects walked and then jogged on the treadmill. Subjects were instructed extensively on safety procedures including getting on and off the treadmill and communicating with the operator. Each subject was asked to practice terminating the test several times to ensure maximum safety.

2) Subjects walked on the treadmill with headgear.

3) Subjects walked and then ran on the treadmill with the headgear and the Rudolph valve in their mouths.

4) Subjects underwent a progressive practice run until voluntary cessation. No motivation was given.

5) The practice protocol may be summarized thus:

Trainees:

Men ran at initial speeds of 6.5 mph and women at 5.5 mph for a warm up period of two minutes. At two minutes the treadmill speed was increased to 7.5 and 6.5 mph for men and women respectively. Thereafter speed remained constant. After two minutes at this work load the treadmill was raised 2% every two minutes until voluntary cessation of the test. The above protocol was designed after extensive familiarization procedures with control and trainee subjects.

High Mileage Runners:

Trained runners underwent a practice run at 7.0 - 7.5 mph and 7.5 - 8.0 mph for women and men respectively.

6) Following the orientation procedures all subjects were asked to return to the laboratory for a pre-programme test on two separate occasions. Repeat tests were used to determine the reliability of the aerobic maximum (VO2 Max) measure, to ensure complete familiarity with the test and to control for learning effects of testing.

Anthropometric Measures:

Height and weight were measured by standardized procedures. Weights were measured on a beam balance scale on a weekly basis. The scale was calibrated against known weights on a daily basis. Percentage body fat was estimated via the following protocol:

1) Subject was weighed in a bathing suit on a beam balance scale having arrived at the laboratory 3 hours after the last meal.

2) The subject was instructed to enter the tank and the chart recorder was calibrated according to standardized procedures.

3) Vital capacity was measured in each subject while seated on a submerged chair.

4) Subjects held their breath and submerged fully for a time sufficient to establish a chart recording. Smooth movements were emphasized.

5) A satisfactory chart recording was accepted when three consecutive measures were within one half of one unit on chart recorder paper.

6) Temperature of the water and density of the water was taken before any measurement. Water temperature was maintained between 32-34 degrees celcius for subject comfort.

7) Body density, percentage body fat and lean body mass were estimated on an equation based on the two compartment model ie: fat and non-fat component (Brozek et al., 1963).

Endocrine Measures:

1. Baseline Investigation of Male Subjects:

A single 5 ml baseline blood sample was obtained from each subject in the study on a pre and post program basis for the assessment of LH, FSH, cortisol, prolactin, total testosterone, sex hormone binding globulin and the free testosterone index. All baseline samples were taken at a standard time of 1600-1800 hours at least three hours after the last meal and at least 24 hours since the last bout of exercise. In addition, blood samples were obtained at 15 minute intervals over six hours for evaluation of LH pulse patterns from six males in the training group and six males in the high mileage running group. To determine mean basal LH and FSH levels, the initial sample from the six hour sampling period was used. Six hour sampling was conducted in a three hour post absorptive state and at least 24 hours after the last bout of exercise. The ingestion of non-caffeine based beverages was allowed during the course of the blood sampling. Any movement was discouraged for control purposes, although bathroom visits were allowed. All blood samples were allowed to clot at room temperature, were centrifuged to separate serum and stored at -20 degrees celcius.

2. Monitoring of Hormones during Training in Male Subjects:

The hormones measured at baseline (total testosterone, LH, FSH, cortisol, prolactin) were also measured at the conclusion of the study. Six hour sampling was repeated at the end of the study in male subjects for examination of spontaneous LH pulse patterns as previously described. LH amplitude, pulse frequency and area under the LH curve were compared to pre-program levels and analyzed according to the cluster analysis method devised by Veldhuis and Johnson(1986) and computerized in the Munroe program in 1987.

Psychological Measurements:

The Eating Attitudes test and The Eating Disorder Inventory (EAT, EDI)

The Eating Attitudes Test originally comprised 40 items and was designed as a self report scale of anorectic behaviours and attitudes towards food (Garfinkel and Garner, 1979). The test has been used to detect anorectic tendencies and eating behaviours in runners (Henry, 1982) and in ballet dancers (Garfinkel and Garner, 1982). The scale has since been modified to a 26 item test comprising a 3 factor scale of anorectic behaviours. The three factors were identified as:

1) Dieting.
2) Bulimia and preoccupation with food.
3) Oral control.

The EAT 26 correlates highly with the EAT 40 (r=0.98). A score of 20 points was taken to represent an anorectic score. This compares with the original version score of 30 points.

The EDI represents a factorized extension of the EAT and was also devised by Garfinkel and Garner (1983). The EDI was completed by all subjects. These inventories were completed on a pre and post program basis by all subjects.

Nutritional Assessment:

1. Methodology.

Nutritional intake was assessed by a 3 day diet diary method. The diary was completed on a thursday, friday and saturday on an alternate weekly basis. The food record diary was developed at the University of Alberta by the Faculty of Dentistry and Pharmacy in association with the Kellogg Company. Diaries were analyzed utilizing a computerized 20,000 item data base and analyzed for exact nutrient content. Nutritional data was entered into a computer by an expert in the area of nutrition and diet diary coding and scoring. All information was treated as highly confidential.

2. Baseline Assessment.

All participants attended a one hour instructional session by group and conducted by a nutritionist. During the session detailed instruction was given on the techniques of recording dietary intake. Food models were used extensively to train accuracy in meal size and portion size estimation. Following the orientation session all subjects were given two diaries for the determination of baseline caloric intake. These books were evaluated in detail by the nutritionist. Any problems arising from the completion of these baseline diaries were dealt with by phone of personal interview. All subjects were informed that they had access to the nutritionist for consultation for the duration of the investigation.

3. Intra-Program Assessment.

Diaries were completed by all subjects on an alternate weekly basis. Each month a randomly selected sample was chosen by the nutritionist for accuracy checks and integrity. Any difficulties or apparent inaccuracies encountered during these checks were followed up by the nutritionist and reported to the investigator.

Summary of Programme Procedures by Group:

High mileage Group.

Following all pre-tests as previously outlined members of this group were instructed to continue training as per their own schedules and to record dietary intake as outlined. Subjects were asked by the nutritionist to exercise the utmost integrity in completing the diaries and were told that the success of the investigation depended on the same. All subjects were asked to record any injury periods. In the event of illness the subjects were instructed to complete diaries on the first thursday to saturday period after normal health resumed.

Training Group:

1. Matching and Grouping:

Following all pre-test and orientation procedures the training group was paired and matched on Cooper 12 minute run performance, VO2 max. and gender. Subjects were randomly assigned to two training groups hereafter referred to as groups A and B. This procedure was followed to permit subjects to choose suitable training times.

2. Programme Outline:

Each training group met with the investigator and the program was discussed according to a standardized agenda. Training progressions were explained on the basis of simple physiological principles. Instruction was given on the purchase of appropriate footwear and an attractive discount offered via an arrangement with a local sports store. This ensured that the trainees were adequately protected in terms of appropriate footwear. Training intensity was determined by a simple target heart rate zone method (200 - age, upper limit and 170 - age lower limit) and the importance of maintenance of training intensity and the heart rate training sensitive zone was explained. Subjects were instructed to monitor heart rate response to exercise on an ongoing basis to ensure that training intensity was maintained. A generalized warm-up and flexibility programme was outlined to the participants to ensure conformity to a standardized program in the event of their being unable to attend training sessions. Warm up was emphasized as a means of reducing the incidence of injury.

At the beginning of the program all subjects were filmed running on the treadmill and on the track in order to assess running style in terms of pronation and supination (varus and valgus alignment) and heel and toe strike. This evaluation was used to offer preliminary advice on the purchase of training footwear and thereby minimize the confusion facing the typical naive running shoe buyer.

The running program was standardized to incorporate a weekly and monthly distance increase of approximately 8 kilometres per week for each month up to six months. The programme was thus designed to increase training distances to between 40 and 56 kilometres per week. Although this was the target distance all trainees were encouraged to self-monitor their progress and to proceed at their own rate should the prescribed program be too severe. This method of self regulation of intensity and volume has been successfully implemented by others in maintaining a high rate of subject compliance and low injury rates (Boyden et al. 1984). All subjects were required to keep a record of their training pace to assist in categorization of effort later in the analysis stage.

3. Weekly Monitoring of Weight.

Trainees were required to attend a weekly weighing session. Weight was measured on a beam balance scale which was recalibrated with known weights on a daily basis. All weights were taken in a "T" shirt and shorts and at least three hours post absorptive. All other subjects were measured in the same manner.

4. Training Times:

Training schedules were discussed with each subject such that all subjects could attend the university at a suitable training time. This allowed the investigator and training assistant to personally supervise all training sessions. Training times were restricted to three periods per day and subjects were encouraged to vary the training time.

5. Training Environment:

To ensure that the effects of training and diet were not confounded by outside temperature, all training took place at the University of Alberta, Van Vliet Centre. The 200 metre track permitted the measurement of precise distances. Outdoor running was actively discouraged although it was recognized that some outdoor running would have to take place when subjects schedules would not permit their attending the University. Some outside runs were arranged by group to offset the monotony problem.

6. Responsibility of the Investigators:

Two trainers trained the groups. Trainers were both at the masters level within the University of Alberta, experienced long distance runners and experts in training prescription. To control for any experimenter bias the trainers met on a daily basis and regularly swapped group responsibilities.

7. Dietary Counselling:

Nutritional advice was limited to that pertaining to the completion of diet diaries. No advice was given on macro-nutrient bias with training or with regard to nutritional supplementation.

Controls.

Controls were selected as previously outlined and underwent measures previously discussed The only requirement of the control subjects was that they kept a faithful record of their caloric intake via the diet diary method and maintain their sedentary lifestyle. Follow-up interviews were conducted by the investigator and the nutritionist to ensure that the utmost integrity was exercised by all the control subjects. Any failure to meet the expectations of the investigators was met with immediate exclusion from the investigation and nullification of the payment contract. It was emphasized at the beginning of the investigation that payment was contingent on the receipt of a full compliment of diaries completed to the satisfaction of the nutritionist.

Design and Analysis:

1. The experiment represents a 3 (group) x 2 (gender) x 3 (time) mixed factorial design. Factor 1 was group; high mileage runner, trainees and control subjects. Factor 2 was gender (male, female) and factor 3 was time; pre mid and post program (0, 12 and 24 weeks).

2. Analysis of data was conducted as follows:

a) UANOVA (unique analysis of variance, Dr. T. Taerum, University of Alberta): A three way anoya with repeated measures was used to examine the effects of training on physiological, hormonal and psychological variables examined in the investigation.

b) UANCOVA (unique analysis of covariance): To examine the effects of initial fitness level on training induced changes in VO2 Max, and to account for the effects of body weight and lean body mass on caloric, fat, carbohydrate and protein intake in men and women the unique analysis of covariance procedure was used. Covariates utilized were pre-VO2 max, pre-weight and pre-lean body mass.

c) INDIVIDUAL SUBJECT INVESTIGATION: To examine the effects of exercise on dietary behaviour, the dietary profiles of trainees were examined. A profile analysis was calculated to establish trends in dietary intake over a six month training period.

d) CORRELATION COEFFICIENTS: To examine the relation between caloric intake, macro-nutrient intake and relative dietary measures (eg: calories/kg body weight and lean body mass) and nutrient intake and hormonl levels, Pearson Product Moment Correlation Coefficients were calculated.

Significance Levels:

The 0.05 alpha level was accepted as the appropriate degree of significance in the investigation throughout all analyses.

Limitations of the Investigation:

1) **Attrition**: A certain degree of attrition must be anticipated associated with a training program such as that proposed. Expected reasons will include unforseen relocation, personal circumstances and injury due to overuse.

2) **Physiological Measurement**: Inherent error in the estimation of percentage body fat from the densitometry technique. Estimation of residual volume.

3) **Nutritional Assessment**: The use of dietary diary techniques although more reliable than the 24 or 48 hour recall methods are susceptible to subject integrity, non-homogeneity of food intake on week days and problems of retrospective dietary completion.

4) **Hormonal Measures:** Error in the utilization of radioimmunoassay techniques for the measurement of hormones. Assay sensitivity and precision of techniques are associated with inter and intra-essay coefficients of variation.

5) **Subject selection:** Due to the nature of the investigation subjects were selected from respondents to three advertisements. This was to ensure a high mileage and sedentary control group and a low risk training group.

Delimitations of the Investigation:

1) 20 high mileage runners; 10 men and 10 women training at least 56 km per week (Hereafter called HMRM and HMRF).

2) 40 active and healthy men (20) and women (20) taking part in a six month endurance running program (Hereafter called TRM and TRF).

3) 30 control/comparison subjects (15 men and 15 women): selection based on a lack of participation in regular physical exercise (Hereafter called CONM and CONF).

4) Endocrine parameters will be measured from blood samples taken at a standard time of day; 1600-1800 hrs and between 1200 and 1900 for six hour serial sampling.

A Definition of Terms.

1) **Habitual Running**: For the purposes of this investigation the habitual runner is a man or woman who trains daily, covers at least 48-56 km per week and who does not necessarily compete. This individual typically has to run daily and will experience considerable guilt and anxiety if a run is missed with the concomitant development of withdrawal-like symptoms if deprived of running altogether such as in the case of injury. Furthermore, such an individual may not cease running even in the event of an injury. Such a runner would also be described as negatively addicted. (Glasser, 1969).

2) **Maximum Oxygen Uptake**: The maximum rate at which the body can utilize oxygen in the breakdown of carbohydrates and fats to C02, H2O and heat, with the concomitant production of ATP. The measure may be expressed in litres per minute (absolute) or as millilitres per kilogram of body weight per minute (relative).

3) **Anorexia Nervosa**: A psychopathological condition of self induced cachexia and starvation characterized by a loss of at least 20% of normal weight, a relentless pursuit of thinness and unceasing fear of obesity and overeating. The anorectic patient suffers from a distorted body image and has been described as engaged in a battle for personal self-control through oral control methods (Bruch, 1974). The syndrome is further complicated by physiological dysfunctions including an abnormal endocrine profile and severe, life threatening electrolytic disturbances (Garner and Garfinkel, 1975;

1978). The prognosis for the disorder is poor with a mortality rate as high as 15% (Dally, 1969).

4) **Activity Anorexia**: A condition generated under laboratory conditions in animals. Activity anorexia occurs in animals (rats) given unlimited access to an activity wheel and subjected to food schedules, typically 60-90 minutes in length. The condition arises from the interaction of opportunity to exercise, daily rate of exercise increase and food schedule and or deprivation. The interaction of the above variables appears to increase activity while food intake declines. The activity increase may suppress food intake which in turn leads to an activity/starvation induced weight loss. If unchecked the process will result in the death of the animal (Epling, Pierce and Stefan, 1983; Epling and Pierce, 1988).

Endocrine Definitions:

1) **Testosterone** (T): Testosterone is a steroid hormone secreted from the interstitial/ Leydig cells of the testes (95%) with 5% being secreted from the adrenal glands. T is released in response to the action of the gonadotropin, Luteinizing hormone (LH) which in turn may be regulated by the action of Prolactin (PRL) in sensitizing the Leydig cells to LH. T has both androgenic and anabolic properties. The hormone functions in the production of the secondary gender characteristics of the male and may be associated with libido and potency. The anabolic action of the hormone lies in the synthesis of proteins from amino acids.

2) **Protein Binding of Testosterone**: In men testosterone circulates in the blood bound to specific and non-specific binding proteins and in free form. The largest portion is bound to sex hormone binding globulin (SHBG)(60%)

with remainder bound to albumin (38% approximately) or in free form (2%). SHBG binds testosterone based on a high affinity low volume basis and albumin binds T on a low affinity high volume basis. Protein binding of T modifies the biological availability of the hormone such that SHBG bound T is unavailable to enter the cell to initiate protein synthetic reactions. Free T and non-specifically bound T most likely represent the biologically available portion of the total circulating hormone (Pardridge, 1981).

3) **Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH)**: FSH and LH are secreted in a pulsatile fashion from the anterior pituitary in response to Gonadotropin Releasing Hormone (GnRH). GnRH mediated pulses occur at approximately 90 minute intervals during the majority of the female menstrual cycle but decrease in frequency during the mid and late luteal phase (to approx. one pulse per 3 hours). In men LH is released in a similar manner to that in women ie. is regulated by the pulsatile release of GnRH from the hypothalamus. LH regulates testosterone production from the Leydig cells. FSH is essential for normal spermatogenesis in the seminiferous tubules. LH pulses occur at approximately 90-120 minute intervals.

In women FSH and LH interact in a complex series of hormonal events during the menstrual cycle.

4) **LH Pulses**: The GnRH mediated episodic release of LH from the anterior pituitary occurs at approximately 90 minute intervals during the follicular phase of the cycle and declines to one pulse per three or four hours in the mid to late luteal phase. Pulse frequency in men is generally lower. The pulsatile release of LH is under control of the hypothalamic peptide, GnRH which is released in a similar pulsatile manner from the arcuate nucleus area of the

median eminence area of the hypothalamus but is principally controlled by the circulating levels of the hormone itself by classic negative feedback loops.

Operational Definitions:

1) VO2 Maximum: The maximum amount and rate of oxygen consumption per minute following an incremental bout of exercise. The maximum oxygen consumption point shall be taken as the maximum value achieved prior to or at voluntary exhaustion. Values will be expressed as an absolute measure (litres/minute) or as a relative measure (ml/kg/minute). This value will be confirmed by a supra-maximal test.

2) Supra-Maximum: A supra-maximal test of aerobic power will be conducted following the VO2 max. test to confirm the true maximum of the subject. The subject shall be allowed to rest following the first test until the heart rate has fallen to approximately 120 beats per minute. At this point the subject shall resume running at the final elevation and velocity achieved during the first test. This test to shall be terminated at voluntary exhaustion (see MacDougall, Wenger and Green, 1982).

3) Percentage Body Fat: The amount of fat as estimated by the under water weighing method assuming a two compartment model of the body (fat and non-fat). The equation for the calculation of body fat is derived from that of Brozek (1963).

4) Dietary Intake: The amount and quality of food intake consumed by the subjects of the investigation as assessed by a three day diet diary (Kellogg/University of Alberta Data Base). Daily caloric intake shall be taken as an average of three consecutive daily dietary intake measures.

5) Eating Attitudes: Eating Attitudes shall be defined as the scores attained on the Eating Disorder Inventory (EDI) (Garner and Garfinkel, 1983), and the EAT 26 (Garner and Garfinkel, 1982).

6) LH Pulse: An LH pulse shall be defined as an episodic release of LH as detected by the cluster analysis method of Veldhuis and Johnson (1986) computerized by Munroe et al. 1987.

Independent and Dependent Variables:

Independent Variables:

Number of kilometres of training completed per week and per total program.

Dependent Variables:

a) Nutritional Intake: Total calories, percentage intake of macro-nutrients.
b) Basal hormone levels (T, LH, FSH, prolactin and cortisol).
c) LH pulse patterns.
d) Percentage body fat.
e) kg fat.
f) Lean body weight (kg).
g) Body weight (kg).
h) EAT scores (Eating Attitudes Inventory, EAT 26)
g) VO2 Maximum (ml/kg/min., liters/min.)

All of the above are described in the methods section.

Chapter 3.

Results

Analysis of Variance:

The overall group (HMR,Trainee, Control), time and gender model was tested via a Three Way Analysis of Variance (UANOVA) programme with repeated measures and missing data capability. Analysis of covariance (UANCOVA) was also employed on certain measures. Results of the UANOVA analysis are reported under the following headings:

1) Subject profiles, attrition and compliance.

2) Training distances achieved and cardiovascular adaptations to a six month training program.

3) Anthropometric changes associated with a six month training program.

4) The effects of a six month training program on dietary intake, macronutrient composition and energy expenditure.

5) Individual subject dietary profiles during a six month training program.

6) Dietary surplus and deficit data.

7) A comparison of dietary intake to the recommended nutrient intake for Canadians.

8) The effects of a six month training program on Eating Attitudes and Eating Disorder Inventory Scores.

9) The effects of a six month training program on reproductive hormones and LH pulsatile release.

10) Relationships among variables.

Results:Section 1: Group Personal Data and Program Compliance and Attrition.

Compliance and attrition rates:

Of the 10 high mileage men and 10 high mileage women who began the investigation, 10 men and 7 women completed the investigation. Three women subjects were lost from the investigation. Two were omitted for non-compliance in dietary recording requirements and one was lost due to injury.

Of the 20 men and women who began the training program, 14 men and 13 women completed the requirements of the investigation. Four subjects (3 women and 1 man) dropped the program due to previous diagnoses of anorexia nervosa. The subjects considered that the program (ie: running and recording diet intake) was arousing old fears about body weight and food consumption. Two men were eliminated from the investigation due to a lack of co-operation in terms of dietary recording. Three men developed knee and lower limb injuries which forced them to retire from the investigation. Of the four women who did not complete the investigation two did not comply with dietary recording requirements whereas two developed knee and lower leg injuries which necessitated their dropping out.

The control group were by far the most difficult group in terms of maintaining interest. This was despite the incentive of financial remuneration for completion of diet diaries. Of the 15 men and 15 women who were selected as controls from the sedentary group interviewed, 11 women and 7 men completed the study requirements. In all cases, failure to complete the investigation was associated with failure to complete the diet diaries to the satisfaction of the nutritionist and not appearing for testing procedures.

Pre Investigation differences in Age, Height and Weight.

HMR were significantly older (39.9 ± 2.9 years of age), shorter (162.7 ±1.45 cms) and lighter (59.91 ± 2.03 kgs) than the TR group (31.2 ± 1.51 years, 173.6 ± 2.05 cms, 69.15 ± 2.26 kgs). HMR were also significantly older than the CON group (26.3 ± 1.07 years). TR were significantly taller but not heavier than control subjects (163.49 ± 1.56).

A complete record of mean height, weight and age of subjects completing the investigation is reported in Table 1. There were no group differences in pre investigation lean body mass (LBM) although there was an expected significant gender difference in LBM ($p<0.0001$).

Table 1. Subject Profiles.

GROUP	AGE x/sem (yrs)	HEIGHT x/sem (cms)	WEIGHT x/sem (kgs)
HMRM	47.2	165.89	65.42
	2.53	1.56	1.8
HMRF	29.4	158.09	52.04
	3.68	1.57	1.54
TRM	32.8	180.86	76.91
	2.44	1.99	2.56
TRF	29.4	165.71	60.80
	1.67	2.11	2.02
CONM	25.6	169.97	73.43
	1.51	1.00	4.95
CONF	26.7	158.79	54.06
	1.53	1.35	1.44

* *Means and standard error of the means are for subjects who completed the investigation. Older subjects were lost from the training group whereas younger subjects were lost from the high mileage runner group; particularly HMRM.*

Results: Section2: Training achievement and cardiovascular fitness changes due to training:

Training distance achieved by HMR and Trainees during the Six Month Program:

Description of Results:

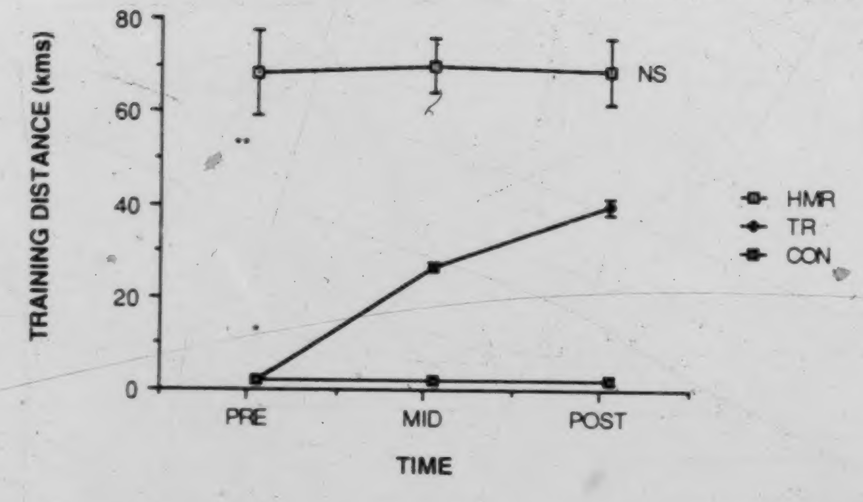
Main Effects:

Group main effect was highly significant (p<0.0001). HMR trained significantly longer distances than the TR group (67.04 ± 6.79 v 20.62 ± 0.77 kms/week). Gender effect was significant (p<0.007). Men trained over greater distances than women (45.78 ±5.9 v 29.9 ±3.69 kms/week). The time main effect was also highly significant (p<0.0001). Training distances increased from 25.67 ±6.03 kms/week to 48.84 ± 3.62 kms/week over the six month period across all groups (see figure 1). The gender training difference was not consistent for both HMR and TR since there was a gender x group interaction. There was an overall significant difference between training volume in HMRM and HMRF but not TRM and TRF. The gender x time interaction was not significant. The time main effect was accounted for by significant increases in training distances in the TRM (pre, 0, mid, 25.1, post, 39.5 km/week) and TRF (pre, 0, mid 23.62, post, 35.1 km/week) groups (p<0.05) but not HMRM, HMRF, CONM and CONF (see table 2). However, although the TRM group and HMRM group differences remained throughout the investigation only a pre and mid point significant difference in training volume was recorded between the HMRF and

TRF groups ($p<0.05$ Scheffe) (see figure 2). The effects of a six month training program therefore removed the group difference between high mileage women and the women training group.

Table 2. Training Achievements: HMR & TR

Group	Training Distance (kms)		
	pre	mid	post
	x/sem	x/sem	x/sem
HMRM	84.2	77.4	77.6
	12.5	7.5	9.9
HMRF	41.0	54.4	51.6
	4.7	7.0	6.3
TRM	0.0	25.1	39.5
	0.0	1.5	2.6
TRF	0.0	23.6	35.3
	0.0	1.1	2.7



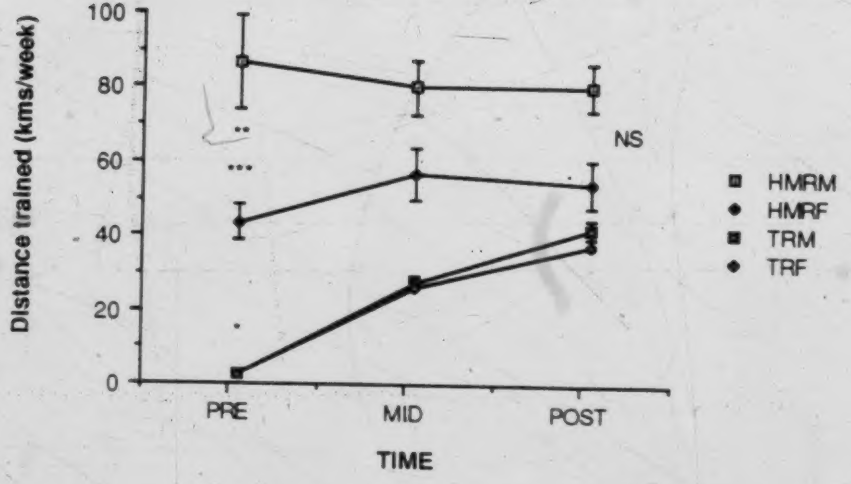
* ** all P<0.05 (Scheffe):

** There was a significant increase in training distance in the TR group from pre to mid, mid to post and from pre to post measurement.*

*** HMR group trained significantly more than TR or CON throughout the experimental period.*

NS: *There was no change in the training distances of the HMR group during the six month experimental period.*

Figure 1. Training distances achieved: HMR, TR & CON by group (mean and SEM)



* ** *** all P<0.05 (Scheffe):

** There was a significant increase in training distance at all stages in the TR group (TRF and TRM)*

*** HMRM trained significantly more than TRM or CONM at all stages.*

**** HMRF trained significantly more than TRF or CONF at all stages except TRF v HMRF at the post measurement period.*

Figure 2. Training distances achieved: HMR & TR: group*gender

The Effects of six months of endurance training on Maximum Oxygen Uptake (VO2 Max.) in HMR, Trainees and Control subjects.

Results: Main effects:

Group ($p<0.0001$), gender ($p<0.0001$) and time ($p<0.001$) main effects were all highly significant. The HMR group had significantly higher VO2 Max. values (57.16 ± 1.91 ml/kg/min) than the TR (44.24 ± 1.15 ml/kg/min) or CON (42.98 ± 1.67 ml/kg/min) groups ($p<0.0001$) although there were no differences between the TR and CON groups. There was a significant gender difference in VO2 Max. (men, 51.51 ± 1.36 ml/kg/min v women, 43.36 ± 1.64 ml/kg/min) ($P<00002$). The time effect was also highly significant ($p<0.001$). VO2 max, increased from 44.33 ± 1.28 ml/kg/min to 49.95 ± 1.23 ml/kg/min. Mean VO2 max. values for all groups are reported in table 3. Results are reported in graphic form in figures 3, 4 and 5.

Group*gender interaction was not significant. The group*time interaction was highly significant ($p<0.001$). Multiple comparisons revealed that HMR maintained a greater aerobic capacity than TR at pre (55.74 ±6.43 v 38.8 ±0.3 ml/kg/min), (mid 56.3 ± 0.51 v 45.8 ± 0.35 ml/kg/min) and post (59.4 ± 0.76 v 47.7 ± 0.34 ml/kg/min) investigation periods and a greater capacity than the CON (CON V02 max. = 41.0 ± 0.31, 42.4 ± 0.53, 42.7 ± 0.51 ml/kg/min, pre, mid, post respectively) group at all stages ($p<0.05$ Scheffe). Surprisingly multiple comparisons revealed that TR did not exhibit significantly higher aerobic power than CON as a result of training. This was due to lower VO2 max. scores in TR as a group than CON at the outset of the investigation.

Training Effects:

There was a significant increase in VO2 max from pre to post measurement period in HMRM (56.1 ± 0.72 v 62.47 ± 0.64 ml/kg/min.) (Scheffe $p<0.05$) (Figure 3). Since weight did not change in this group and training volume did not increase an increase in exercise intensity must be assumed. The use of interval type training for race fitness would account for this increase. Also pre V02 max. in 3 HMRM was low possibly due to detraining due to previous injury. There were no significant changes in HMRF from pre to mid, mid to post or from pre to post training period. HMRM and HMRF maintained greater aerobic power than TR or CON throughout the investigation (see figure 4)

There was a significant increase in VO2 max. in TRM from pre to mid measure (42.47 ± 0.63 to 49.4 ± 0.52 ml/kg/min), from mid to post (49.9 ± 0.52 to 51.8 ± 0.49 ml/kg/min.) and from pre to post (42.47 ± 0.63 to 51.8 ± 0.49 ml/kg/min.) ($p<0.05$, Scheffe).

There were similar significant increases in women on all three comparisons (see table 3) (all Scheffe $p<0.05$). However, the TRF group did not demonstrate greater relative aerobic power than the CONF group at the end of the investigation.

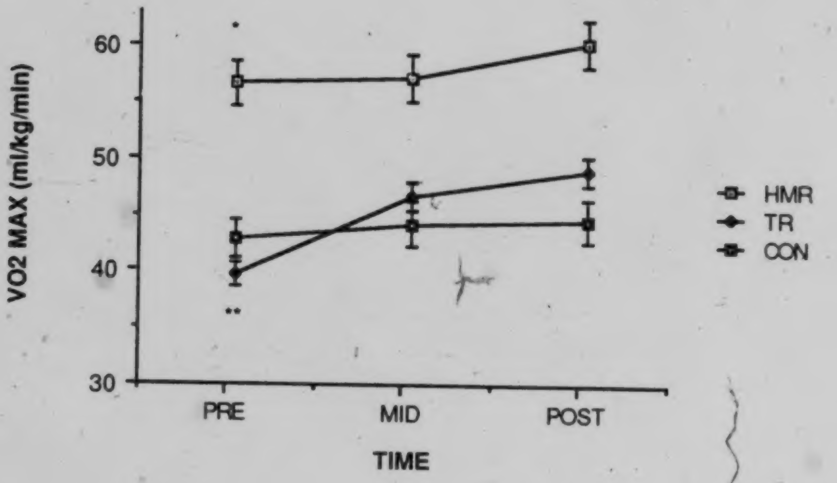
There were no significant changes in the VO2 max. of either CONM or CONF over the training period (see figures 3,4 and 5, table 3). HMRM and HMRF maintained a higher mean VO2 max. than this group throughout. Although CONM had a higher VO2 at the onset of the investigation than TRM (46.7 ± 1.9 v 42.5 ± 1.2 ml/kg/min), CONM exhibited lower mean aerobic power than the TRM group (48.9 ± 1.7 v 51.8 ± 1.3 ml/kg/min) at the end of the investigation.

This difference was, however, not significant. TRF had significantly lower aerobic power than CONF at the beginning of the investigation ($p<0.05$). Although TRF increased their VO2 max (from 35.5 ± 1.2 to 44.1 ± 1.6 ml/kg/min). and were running a mean of 35 kilometres per week at the end of the investigation, the VO2 max levels were not significantly different from those of the CONF group (44.1 ± 1.6 TRF post v 38.9 ± 2.1 ml/kg/min. CONF post). This effect was due to selection of subjects and group bias since randomized design was not feasible in this investigation. Similarly the significantly greater VO2 max. levels of the HMR group were to be expected since this group was selected as a training control group.

Since pre VO2 levels were significantly different at the beginning of the investigation (HMR, 55.77 ± 1.96, TR, 38.83 ± 1.12, CON, 42.03 ± 1.69 m/kg/minute) an analysis of covariance was computed to account for the original group differences. When the effects of the covariate were removed the main effects analysis remained significant (see appendix 9: table 6).

Table 3. The Effects of training on VO2 Max.

Group	Pre X sem	Mid X sem (mls/kg/min.)	Post X sem
HMRM	56.1	57.6	62.5
	3.13	2.87	2.23
HMRF	55.3	54.4	55.0
	1.91	3.12	3.20
TRM	42.5	49.9	51.8
	1.2	1.46	1.28
TRF	34.5	41.2	44.1
	1.21	1.42	1.64
CONM	46.7	48.6	48.9
	1.88	1.98	1.70
CONF	37.9	38.7	38.9
	1.71	2.41	2.10

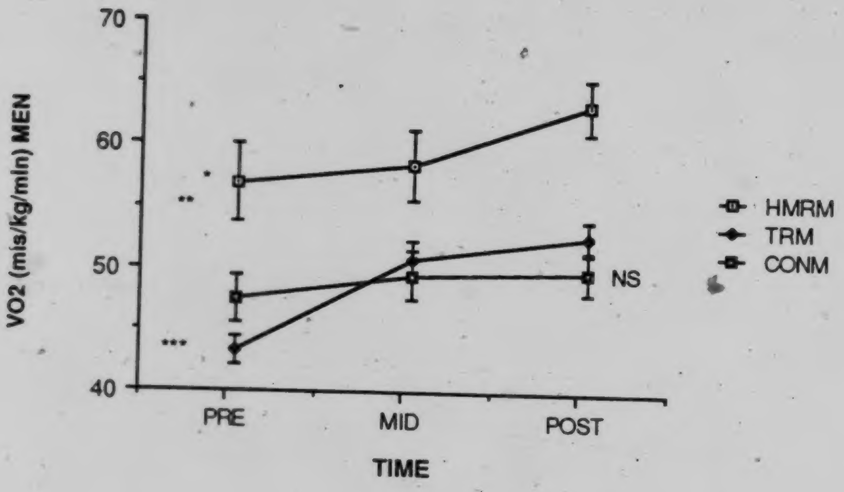


* ** all P<0.05 (Scheffe):

** A significant increase in HMR from pre to post measurement period.*

*** A significant increase in VO2 from pre to post and pre to mid measure in the TR group.*

Figure 3. Effects of training on VO2 max. by group.



* ** ***all P<0.05 (Scheffe):

** HMRM increased from pre to post measure.*

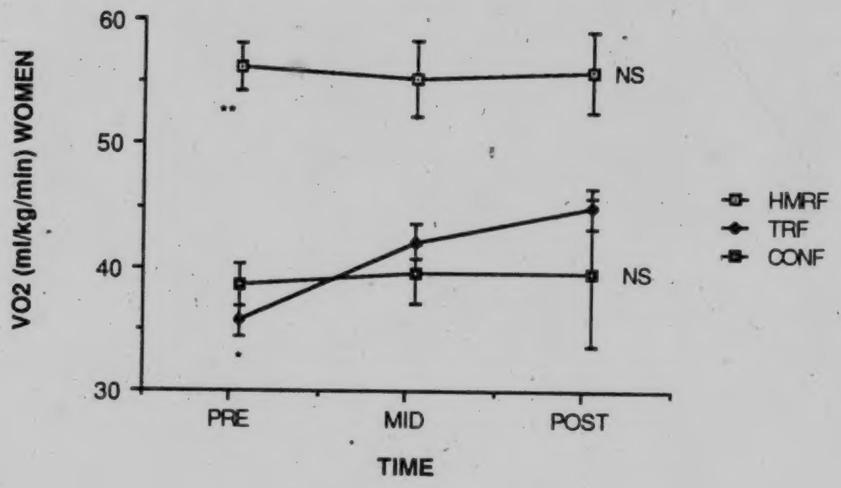
*** HMRM maintained a significantly greater VO2 max. than TRM or CONM throughout the experimental period.*

**** VO2 max. increased TRM significantly from pre to mid and from pre to post measure.*

Although TRM had a greater VO2 max. than CONM at the end of the investigation the difference was not significant.

NS *There was no change in the CONM group.*

Figure 4. Effects of training on VO2 max: group*gender (men)



* ** all $P < 0.05$ (Scheffe):

** TRF significantly increase in V02 max. from pre to mid and from pre to post measure.*

*** HMRF maintained a greater VO2 max. than TRF or CONF throughout the investigation period.*

NS *There was no significant change in the CONF group or HMRF group. Although TRF achieved higher VO2 max. levels than CONF at the end of the investigation the difference was not significant.*

Figure 5. Effects of training on VO2 max: group * gender (women)

Results: Section 3:The Effects of six months of endurance training on anthropometric variables: Body weight, body fat and lean body mass.

Changes in body weight during a six month training program:

Main Effects:

Group ($p<0.01$), gender ($p<0.0001$) and time ($p<0.001$) main effects were highly significant. HMR (59.75 ± 2.05 kgs) and TR (68.25 ± 2.16 kgs) groups differed significantly in weight ($p<0.05$) although TR and CON (62.43 ± 3.26 kgs) and HMR and CON did not differ. Men (71.69 ± 1.65 kgs) were consistently heavier than women (56.35 ± 1.25 kgs) ($p<0.0001$). This was consistent across all groups as revealed by gender*time Scheffe multiple comparisons (see table 4). There was significant reduction of body weight over time from 64.8 ± 1.54 to 63.63 ± 1.39 kgs. ($p<0.0001$). The time effect was due to a significant weight reduction in the TR group from pre to mid, mid to post and pre to post measurement (see figure 7). There were no changes in the other groups. Post hoc Scheffe multiple comparisons revealed that HMRM were not significantly different in weight from CONM.

Effects of training:

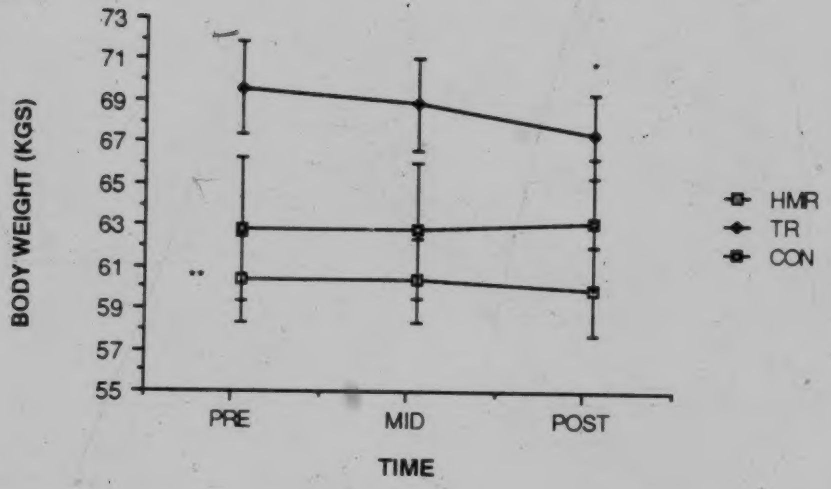
Neither HMRM or HMRF changed weight significantly during the training period (see figures 6 and 7).

There was a significant reduction in body weight from pre to post measure in the TRM group ($p<0.05$) (see table 4, figure 6). However, there was no effect of training on the body weight of the women runners. TRM experienced a significant loss in body weight (79.9 ± 0.37 to 74.02 ± 0.39 kgs, $p<0.05$) following 6 months of running training whereas there was no effect of training on the TRF group (60.8 ± 0.29 to 59.18 ± 0.38 kgs) (see figure 7). TR remained heavier than HMR throughout the investigation but as a group and not by gender.

There were no significant alterations in the body weight of the control group during the course of the investigation (see figure 6).

Table 4. The Effects of training on body weight: means & sem (Kgs).

Group	Pre x sem	Mid x sem	Post x sem
HMRM	65.24	65.24	65.07
	1.80	1.88	2.04
HMRF	52.04	52.54	51.46
	1.54	1.48	1.40
TRM	76.9	76.01	74.02
	2.56	2.39	2.09
TRF	60.8	60.14	59.19
	2.02	2.08	2.02
CONM	73.43	72.93	73.26
	4.95	4.75	3.87
CONF	54.06	54.33	54.68
	1.44	1.45	1.31

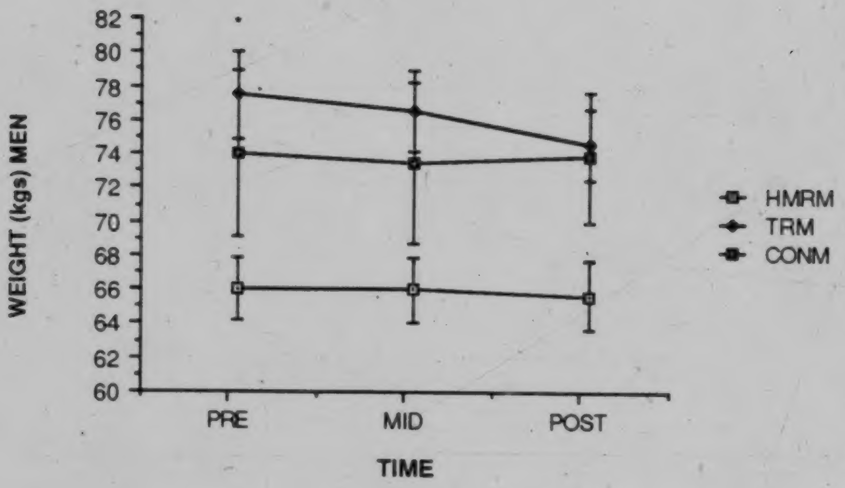


* ** all P<0.05:

** There was a significant reduction in body weight in the TR group.*

*** HMR were significantly lighter than TR or CON throughout the investigation.*

Figure 6. Effects of training by group.



* all P<0.05 (Scheffe): *Only TRM significantly altered in body weight during the investigation.*

Figure 7. Effects of training: group*gender (men)

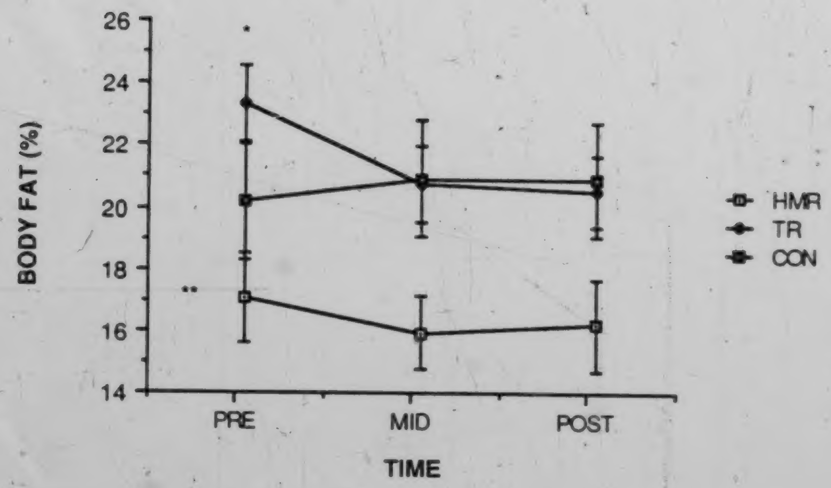
The Effects of training on Percent Body fat and Lean Body Mass.

Main Effects:

There was a significant group ($p<0.001$), gender ($p<0.00001$) and time ($p<0.01$) main effect for percent body fat among the groups. HMR (16.08 ± 1.31%) had significantly lower body fat than CON (20.37 ± 1.85%) and TR (21.19 ± 1.12%)($P<0.05$ Scheffe) although there was no difference between the TR and CON groups. The group differences in body fat were accounted for by significantly lower body fat in HMRF (17.79 ± 1.58%) than TRF (26.2 ± 0.87%) and CONF (24.9 ± 1.68%) ($p<0.05$) but not between HMRM, TRM and CONM. (see figure 9). Men (15.32 ± 0.82%) had significantly less body fat than women (22.93 ± 0.92 %) ($P<0.00001$). Percent body fat decreased from 19.76 ± 0.9% to 18.8 + 0.84 % across all groups ($p<0.01$). Group*gender multiple comparisons revealed that gender differences between TRM (16.74 ± 0.96%) and TRF (26.11 ± 0.87%) and CONM (14.32 + 1.61%) and CONF (24.9 + 1.68%) were significant although there was no significant difference between HMRM (14.89 ± 1.89%) and HMRF (17.79 ± 1.58%). Group*time ($p<0.01$) effect is accounted for by a significant reduction in percent body fat in the TR group from pre (23.03 ± 0.52%) to mid (20.42 ± 0.37%) measure and from pre (23.03 ± 0.52%) to post (20.07 ± 0.35) measure ($p<0.05$) but not mid to post measure (see figure 8). The effect of training on percent body fat was significant in TR as a group only and not by gender. Neither HMR nor CON altered in percent body fat during the investigation. Mean values for body for all groups are reported in table 5. Graphic representations of changes in body fat are represented in figures 8 and 9. As expected there was a drop in body fat in the TR group but not the chronic runners and control subjects.

Table 5. Percent Body Fat in HMR, TR and CON subjects (%).

Group	pre X sem	mid X sem	post X sem
HMRM	15.64	14.80	14.23
	1.88	1.84	2.12
HMRF	18.34	16.79	18.23
	2.27	1.88	1.65
TRM	18.90	15.73	15.60
	1.44	1.06	0.91
TRF	27.47	25.50	25.32
	1.13	1.02	0.94
CONM	13.87	14.50	14.60
	1.41	1.94	1.70
CONF	24.34	25.20	25.17
	1.94	1.56	1.59

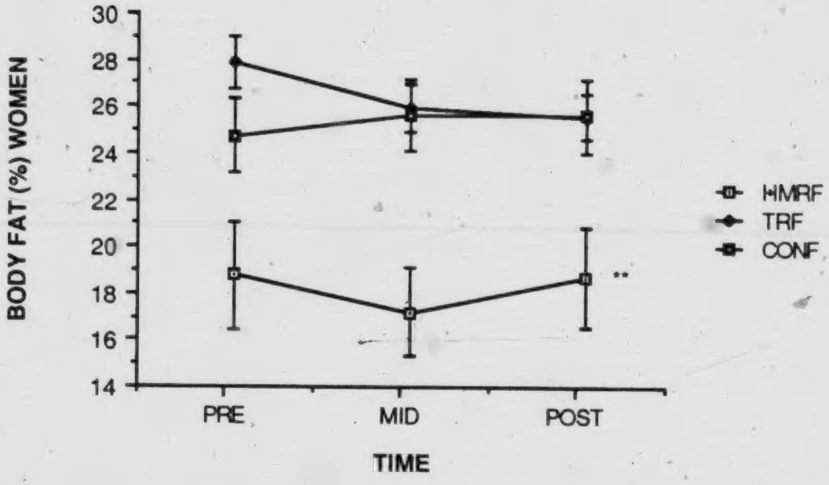


* ** all P<0.05 (scheffe):

** There was a significant reduction in body fat in the TR group with training (not by gender).*

*** HMR maintained less body fat than TR or CON throughout the investigation.*

Figure 8. Effects of training on body fat by group.



** P<0.05(Scheffe): *differences in HMR body fat were accounted for by significantly lower fat in HMRF v TRF or CONF.*

Figure 9. Group differences in body fat in women subjects.

The Effects of training on Lean body mass:

Main Effects:

The group and time main effects were not significant for LBM although there was a significant gender difference in LBM ($p<0.0001$). Men (60.68 ± 1.45 kgs/lbm) had significantly greater lean body mass than Women (42.72 ± 0.9 kgs/lbm). Post hoc comparisons (Scheffe) revealed gender differences between HMRM and HMRF, TRM and TRF and CONM and CONF in lean body mass throughout the investigation ($p<0.05$) (see table 6). Training did not result in any alteration in lean body mass.

Table 6. Mean Lean Body Mass in HMR,TR and CON (Kgs)

Group	pre X sem	mid X sem	post X sem
HMRM	56.1	56.0	56.7
	2.12	2.02	2.07
HMRF	42.2	43.2	41.9
	1.44	1.16	1.17
TRM	63.4	63.9	62.3
	2.38	2.21	2.33
TRF	43.6	44.7	44.8
	1.58	1.61	1.77
CONM	62.5	61.9	62.4
	4.01	4.03	3.63
CONF	40.8	40.5	39.8
	0.86	0.86	1.86

Results: Section 4. The effects of training on caloric intake and dietary composition of HMR, TR and CON.

Analysis of Variance and Covariance:

To control for pre investigation gender differences in body weight and gender differences in lean body mass among the groups an analysis of covariance (Uancova) was computed utilizing weight and lean body mass as covariates in addition to an analysis of variance.

Main Effects:

Caloric Intake

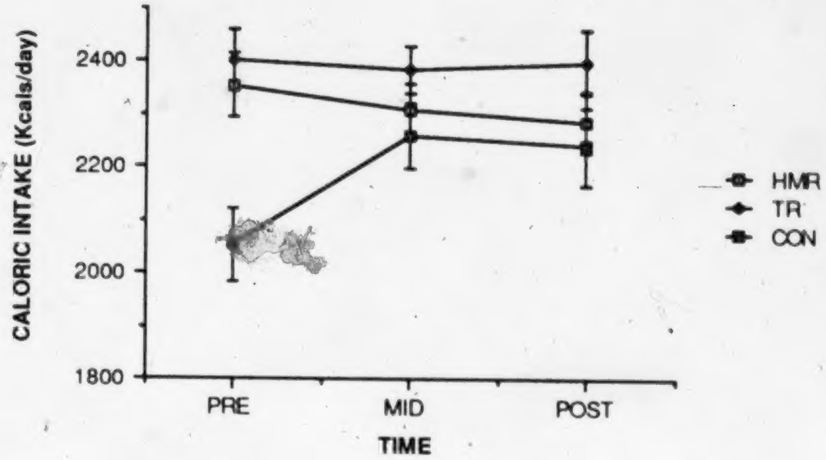
Despite the disparity in activity levels among the groups it was remarkable that there were no differences in group or time main effects. There was a significant gender effect ($p<0.001$). Men (2645.9 ± 79.34 kcals/day) ate significantly more calories than women (1984.5 ± 74.53 kcals/day) (see table 7). No other interactions were significant. Post hoc Scheffe multiple comparisons demonstrated a significant difference in caloric intake between TRM (2792.9 ± 92.9 kcals/day) and TRF (1966.5 ± 111.5 kcals/day) and CONM (2659.9 ± 187 kcals/day) and CONF (1896.2 ± 146.7 kcals/day) (all $p<0.05$). However, HMRM did not differ in the number of calories consumed from HMRF.

The use of weight as the covariate reduced the probability level of significance of gender difference in caloric intake (from $p<0.0001$ to $p<0.01$) and when lean body mass was entered as a covariate in the analysis the gender difference in caloric intake was removed ($p<0.0001$ to $p<0.1$). Differences in caloric intake between TRM and TRF and CONM and CONF were removed when subjects were equated for weight and lean body mass. Mean caloric intake by group and gender is reported in summary in table 7. Group measures of dietary intake are illustrated in figure 10.

Training had no effect on caloric intake in either HMR or TR groups. There was no seasonal change in caloric intake in the CON group.

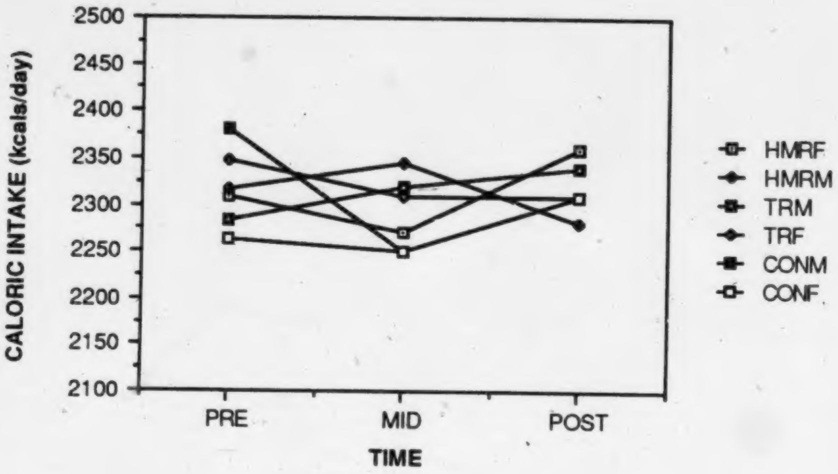
Table 7. Effects of training on caloric intake (Kcals/day).

Group	pre X sem	mid X sem	post X sem
HMRM	2440.5	2446.7	2394.0
	169.5	193.7	215.8
HMRF	2230.7	2111.9	2127.1
	132.9	77.0	192.6
TRM	2750.7	2802.9	2825.1
	105.6	128.3	117.5
TRF	2028.0	1932.3	1939.0
	154.9	149.5	111.4
CONM	2551.2	2733.3	2695.3
	172.1	271.4	184.9
CONF	1780.5	1957.8	1950.5
	123.5	188.7	187.4



There were no significant effects of time or training on the caloric intake of HMR, TR or CON.

Figure 10. Effects of training on caloric intake HMR, TR and CON



When gender was equated with lean body mass the gender effect was removed.

Figure 11. Gender differences in caloric intake

The effects of training on carbohydrate intake in HMR, TR and CON subjects.

Main Effects:

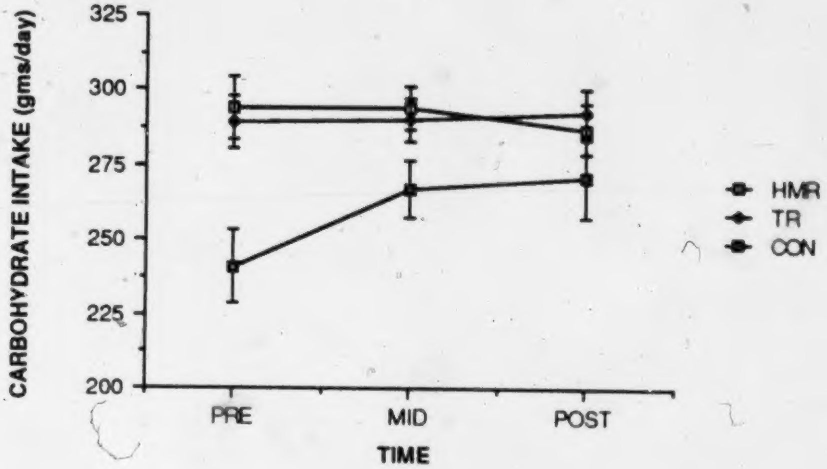
Only gender ($p<0.0001$) and group*gender interaction ($p<0.01$) effects were significant. There was no effect of time or training on the consumption of carbohydrates among the groups. Group*gender multiple comparisons showed that TRM (344.2 ± 19.24 gms/day) differed significantly from TRF (233.3 ±18.35 gms/day) and CONF (230.0 v ±16.4 gms/day) in carbohydrate consumption (all $p<0.05$ Scheffe). When body weight and lean body mass were used as a covariates for carbohydrate intake the gender effect was removed. The group*gender interaction effect remained marginally significant when weight was used as the covariate and was removed when lean body mass was used as the covariate. Group*gender multiple comparisons revealed that when weight and lean body mass were used as covariates only TRM (344.2 ± 19.2 gms/cho) and TRF (233.4 ± 18.35 gms/cho) exhibited any differences in carbohydrate intake. Mean carbohydrate intake by group and gender over time is reported in table 8. Graphic representation of carbohydrate is reported in figures 12 and 13.

Training Effects:

Training had no effect on carbohydrate intake in either HMR or TR There was no time (seasonal) effect on carbohydrate intake in CON

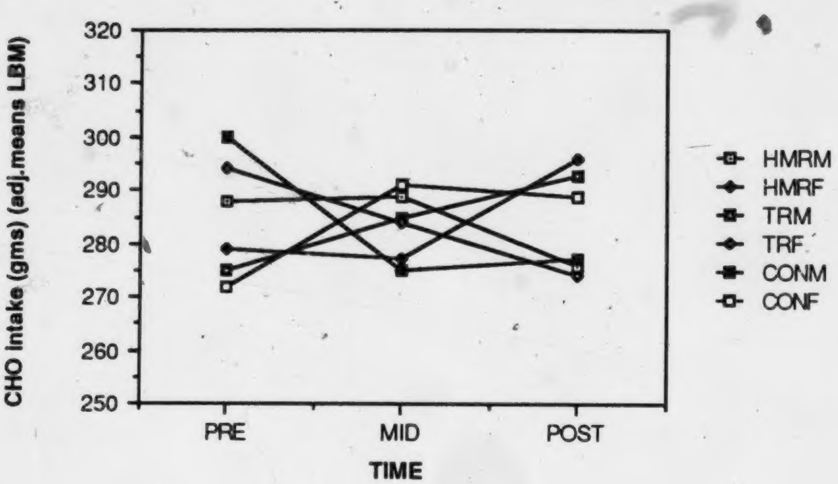
Table 8. Carbohydrate Intake in HMR, TR and CON (gms).

		TIME	
Group	pre X sem	mid X sem	post X sem
HMRM	289.0	299.8	285.4
	23.5	27.5	28.9
HMRF	302.5	287.4	291.6
	14.7	14.8	21.9
TRM	325.0	348.3	359.3
	21.4	21.8	20.7
TRF	250.7	227.7	221.4
	25.9	21.4	19.1
CONM	299.2	311.3	315.7
	45.3	31.3	33.7
CONF	209.7	239.1	243.9
	16.3	26.4	19.6



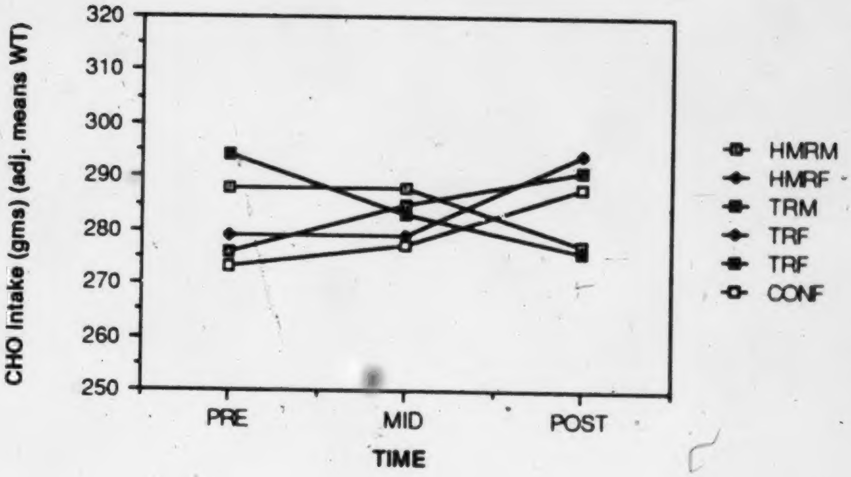
There was no change in carbohydrate intake with training or over time in any of the groups.

Figure 12. Effects of training on carbohydrate intake in HMR, TR and CON



When LBM was equated with dietary carbohydrate intake the gender difference was removed.

Figure 13a: Gender differences in carbohydrate intake in HMR, TR and CON.



A significant gender effect for carbohydrate intake was removed when body weight was used as a covariate in the analysis.

Figure 13b: Gender differences in carbohydrate intake in HMR, TR and CON

The effects of training on dietary fat intake in HMR, TR and CON subjects.

Main Effects:

There were no significant differences in consumption of dietary fat either among the groups or over time (ie: with training). There was a significant difference between genders for dietary fat intake ($P<0.00011$). Men (98.05 ± 4.17 gms/day) consumed more dietary fat than women (73.13 ± 3.75 gms/day). Men ate more fat than women at the pre (100.12 ± 5.1 v 74.37 ± 3.95 gms/day) and mid (99.10 ± 4.33 v 74.47 ± 4.1 gms/day) but not post period of the investigation in all groups ($P<0.05$ Scheffe). The use of body weight and lean body mass as covariates in the UANOVA analysis removed the gender difference in dietary fat consumption.

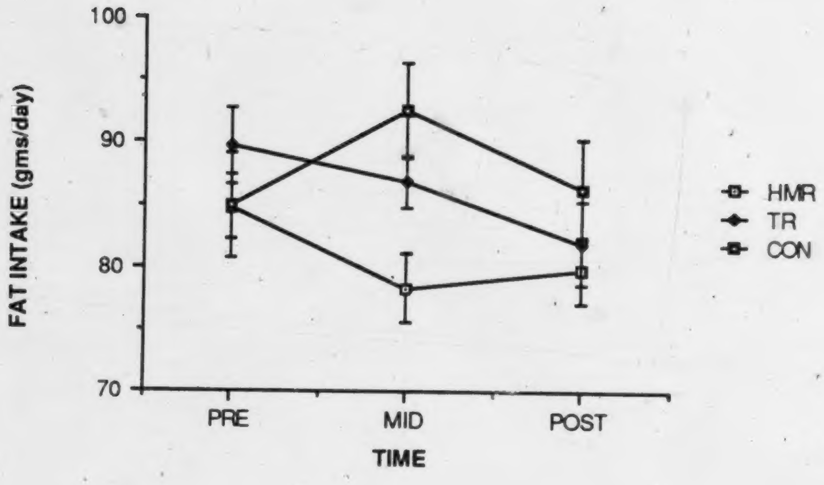
Mean dietary fat intake before, during and after the investigation is reported in table 9. Graphic representation of the results is reported in figures 14 and 15.

Effects of training:

Training had no effect on dietary fat intake in either HMR or TR. There was no seasonal effect on fat intake in the CON group.

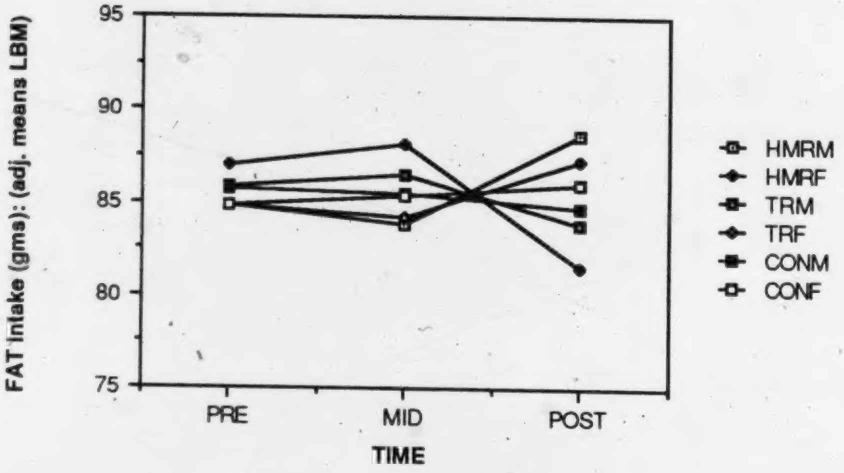
Table 9. Dietary fat intake (gms):

	TIME		
Group	pre X sem	mid X sem	post X sem
HMRM	91.3	81.9	89.7
	7.6	8.2	10.4
HMRF	75.6	73.0	65.6
	6.6	7.2	7.2
TRM	105.2	100.4	93.5
	6.8	7.9	7.3
TRF	73.0	72.3	69.5
	8.1	7.1	5.9
CONM	103.8	115.0	101.6
	11.3	16.9	10.9
CONF	74.6	78.2	76.6
	7.7	9.6	10.6



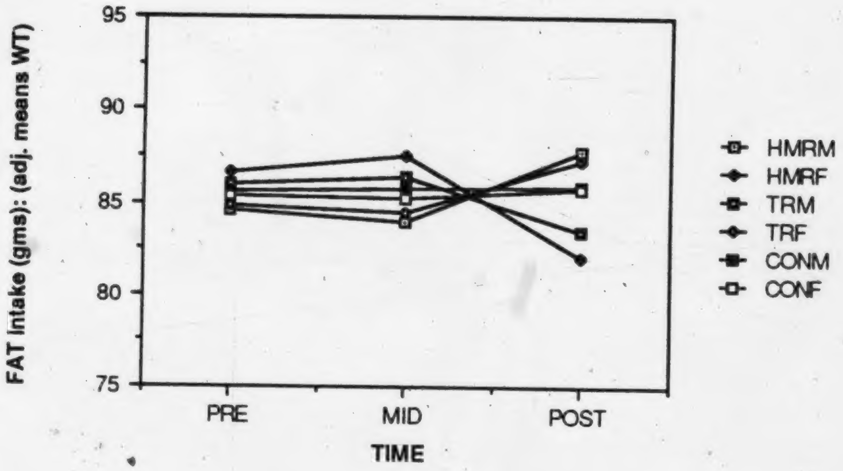
There were no differences in dietary fat intake following six months of endurance running training.

Figure 14. Effects of training on the dietary fat intake HMR, TR and CON.



*Group and group*gender differences in dietary fat intake were removed when LBM was used as a covariate in the analysis.*

Figure 15a: Gender effects: Dietary fat intake in HMR, TR and CON.



Gender and group gender differences in dietary fat intake were removed when body weight was used as a covariate in the analysis.*

Figure 15b: Gender differences in dietary fat intake.

The Effects of training on dietary protein intake in HMR, TR and CON subjects.

Main Effects:

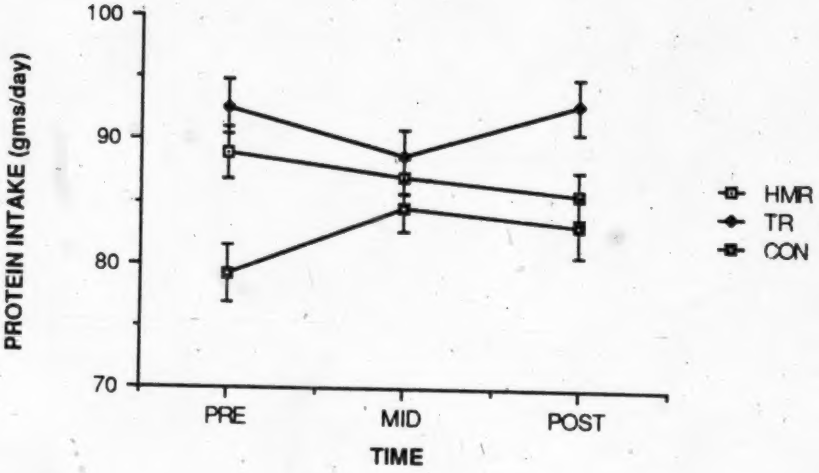
There were no significant differences either between the groups or over time in dietary protein intake. There was a significant difference in dietary protein intake between men (100.75 ± 3.35 gms/day) and women (88.1 ± 2.97 gms/day)($P<0.0001$). This was not consistent over time. Men only ate significantly more protein than the women at the mid and post measurement stages and not on the pre measure ($P<0.05$ Scheffe) There was also a significant group* gender interaction ($P<0.05$). Scheffe post hoc comparisons revealed that only TRM and CONM ate more than TRF and CONF. HMRM and HMRF did not consume different amounts of dietary protein (see table 10). The addition of body weight and lean body mass as covariates in the analysis removed the group*gender interaction effect although the gender effect remained intact when body weight was entered as a covariate. The use of covariates removed the CONF and CONM protein intake difference although the TRM v TRF and CONF differences remained significant. Mean dietary protein intake before, during and after the investigation period is reported in table 10. Graphic representation of dietary protein intake is reported in figures 16 and 17.

Training Effects:

There was no effect of training on dietary intake in protein in either TR or HMR. There was no seasonal effect on protein intake in the CON group.

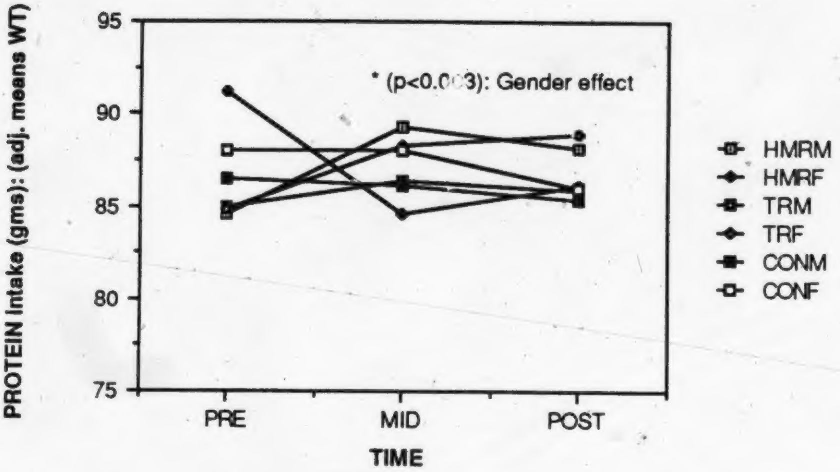
Table 10. Dietary Protein Intake (gms).

	TIME		
Group	pre X sem	mid X sem	post X sem
HMRM	90.0	91.8	90.0
	5.1	5.3	6.8
HMRF	87.4	80.6	79.4
	6.5	3.3	7.1
TRM	111.5	103.1	107.3
	5.2	5.8	3.9
TRF	72.3	73.5	77.5
	2.3	5.5	4.4
CONM	97.2	102.1	106.7
	8.9	10.2	9.2
CONF	69.5	73.4	68.3
	5.2	7.2	9.3



There were no significant differences in dietary protein intake among the groups or over time.

Figure16. Effects of training on dietary protein intake HMR, TR and CON.



There were significant gender differences in dietary protein intake. The gender effect remained intact when body weight was used as a covariate. Analysis of covariance in which lean body mass was used as a covariate removed the gender difference although the p value was P<0.054.

Figure 17: Gender differences in dietary protein intake.

Individual dietary profiles among TRM & TRF.

Since there were no changes with dietary intake over time in the TR group, an individual caloric intake/training profile was examined. Ten TRM and nine TRF were selected from the subjects who successfully completed the investigation. Successful completion of the investigation was determined by a fully completed set of diet diaries and training at least 25 km per week Individual dietary and training profiles were constructed and the mean training distances and dietary profile is shown below in figures 18 and 19. Individual data is reported below with descriptions in figures 18a to 18j and 19a to 19i.

Graph Legends: For all graphs light lines with boxes represent caloric intake; diamonds on black lines represent mean weekly training distance.

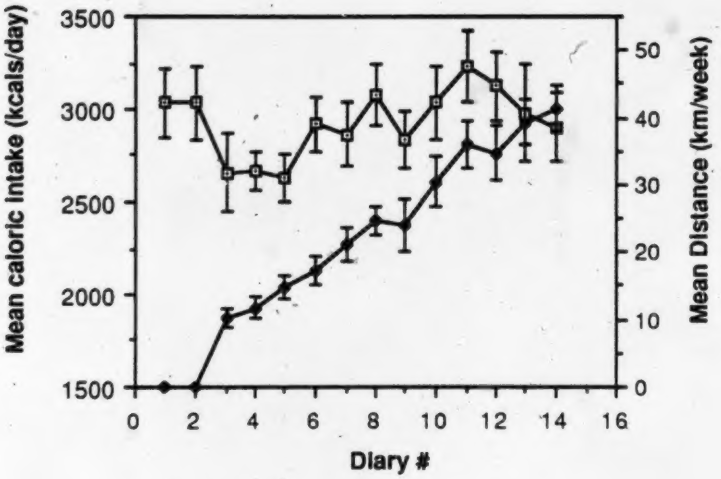


Figure 18. Mean Caloric Intake in 10 TRM during a six month training program.

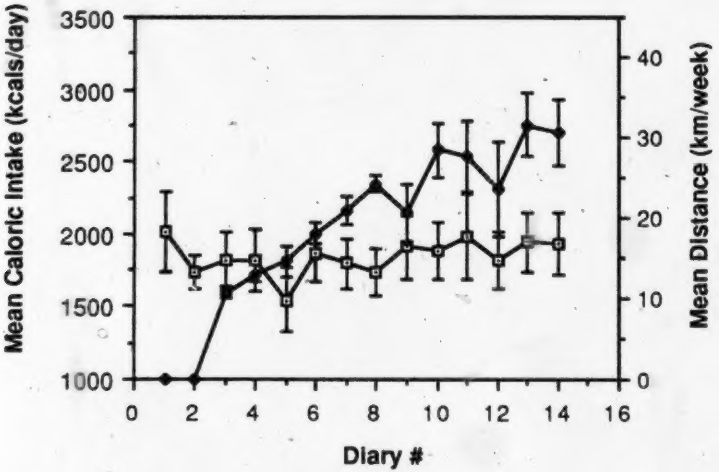


Figure 19. Mean Caloric Intake in 9 TRF during a six month training program,

Description of dietary and training profiles of TRM.

There was a linear increase in distance trained per week over the six month training period in the 10 men selected for individual profiling. Although there was no significant trend in the dietary profile, the caloric intake exhibited a fluctuating pattern which is best described as triphasic :

a) An initial drop in caloric intake at the onset of exercise.

b) A recovery phase characterized by a linear increase to above baseline levels.

c) A secondary decrease phase characterized by a persistent fall in caloric intake despite a linear increase in distance trained by the TRM group.

There was an initial drop in caloric intake in the TRM group during the first 6 weeks of the investigation from a baseline caloric intake of 3035.5 ±193.07 to 2627.6 ± 123.4 Kilocalories per day despite an increase in weekly training of 14.9 ±1.9 km/week. This represented a 13.5% decrease in caloric intake. At week 6 a recovery phase began which appeared to be linear with distance trained. This recovery lasted until week 18 of the investigation. Caloric intake increased to 3237.7 ± 195.7 kilocalories per day which represented an increase of 6.5% over baseline levels. However, after week 18 there was a persistent fall in caloric intake despite an increase in weekly training distance which lasted to the end of the investigation. At week 24 of the running program the TRM group was consuming a mean caloric intake of 2903.3 ± 189.2 kilocalories per day which represented an overall decrease in caloric intake of 4.3% from baseline despite a linear increase in training distance to approximately 40 km per week (25 miles).

Description of Dietary and Training Profiles of TRF.

The dietary profile of the women subjects is remarkable in that despite a linear increase in weekly training distance there was no significant alteration in dietary intake. Caloric intake essentially remains constant throughout the course of the investigation.

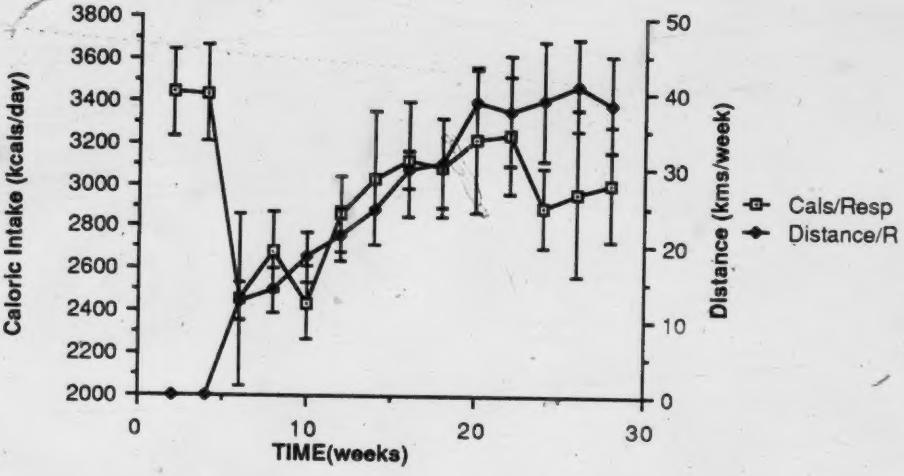
Profile Analysis:

To examine the effects of training on caloric intake in men and women an anova procedure for repeated measures and trend analysis was computed (SPSS 3.0 Time Series and Forecasting). Linear, quadratic, cubic and quartic trends in the dietary profile were examined. Although there was a highly significant overall difference between men and women ($p<0.00001$) no distinct trend in the dietary profile of either men or women was detected. The drop in caloric intake in the TRM group appeared to be distinctive in only 5 of the TRM. In the other 5 there did not appear to be a dietary response pattern. Subsequently a trend analysis was computed for a group, hereafter to be called responders (initial drop in caloric intake)($n=5$) and non-responders (no drop in caloric intake)($n=5$). There was a significant quadratic trend ($p<0.045$) and quartic trend ($p<0.02$) in the responder group and an overall difference in caloric intake between the two sub-groups.

The responder group exhibited a distinct drop in caloric intake immediately after the onset of the investigation which is maintained for a period of 6 weeks. Thereafter, there was an increase in caloric intake although not to baseline levels.

The mean dietary intake and training profiles for the responder and non-responder sub-groups of TRM are reported below in figures 20 and 21.

Since the activity anorexia model assumes a drop in caloric intake associated with exercise intensity and rate of change of daily activity, the rate of change of daily activity was examined via regression analysis. The slope constants for the whole investigation were not significantly different (b = 3.15 ±0.53, responders v 3.19 ±0.14, Non-responders, $p<0.944$). Analysis of the slopes for the first half of the investigation revealed a significantly greater rate of increase in training distance in the responder group (b= 4.211 ± 0.46 v 2.626 ± 0.115, $p<0.011$). The rate of increase in training distance as indicated by the slope constant was 85% greater in the responder group. This suggested a role of rate of change in daily activity in the dietary response of the TRM responder group to training.



There was a significant Quadratic ($p<0.045$) and Quartic ($p<0.022$) trend for dietary intake in the responder group. The slope constant for training distance was significantly greater in the responder group ($b= 4.211$ v 2.63, $p<0.011$) for the first half of the investigation.

Figure 20: Dietary and training profiles for 5 TRM responders.

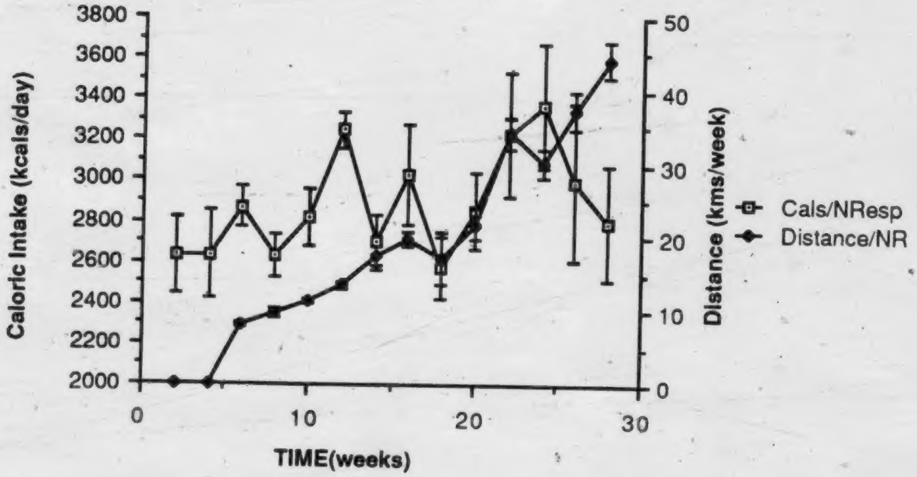


Figure 21: Dietary and training profiles for 5 TRM non-responders.

Individual Dietary and Training Profiles of TRM.

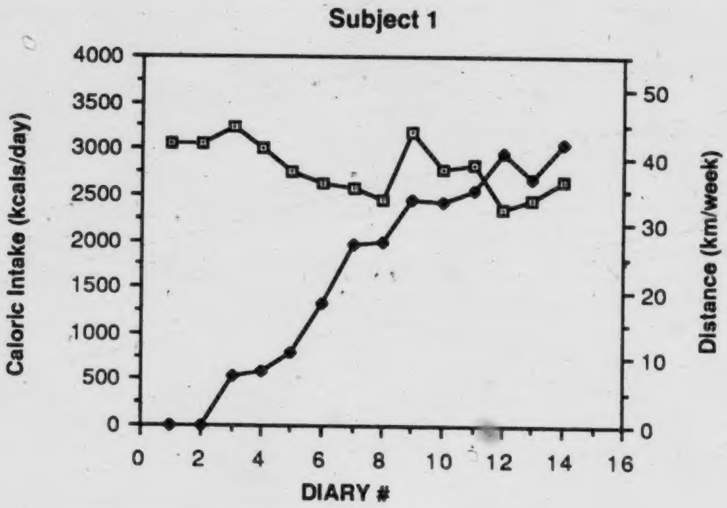


Figure 18a

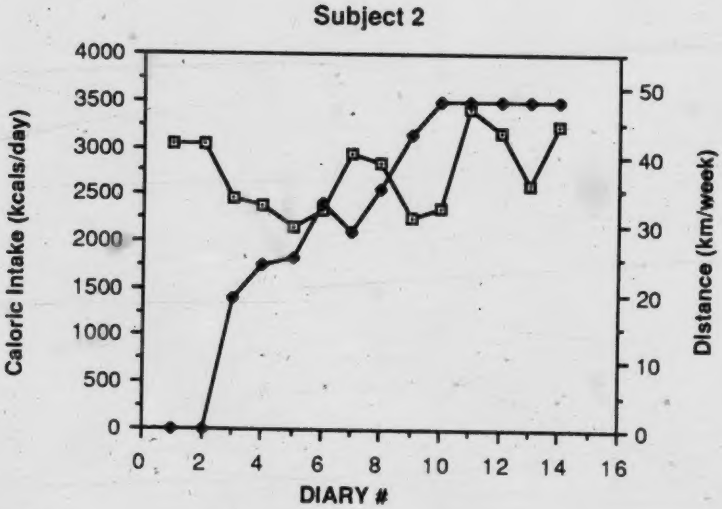


Figure 18b.

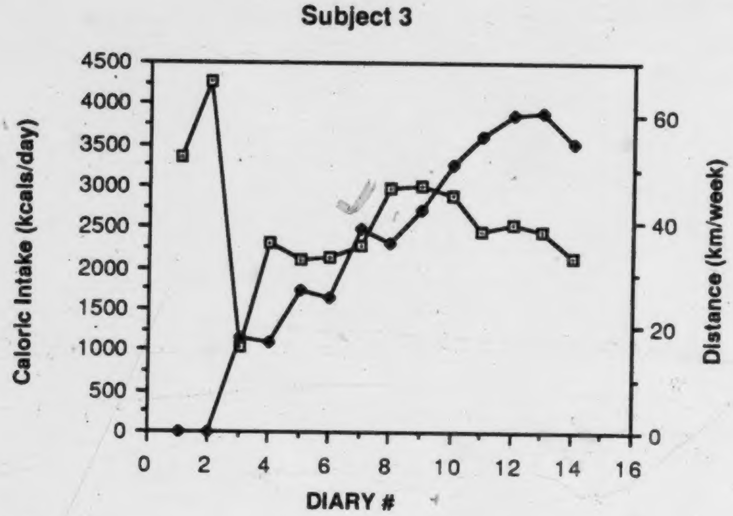


Figure 18c.

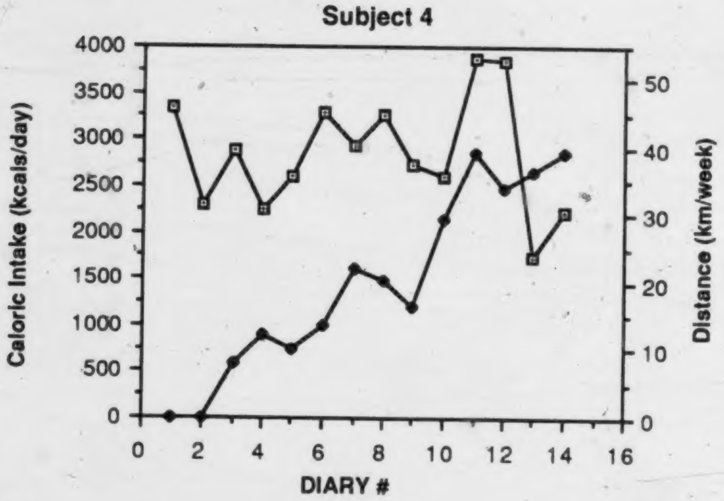


Figure 18d.

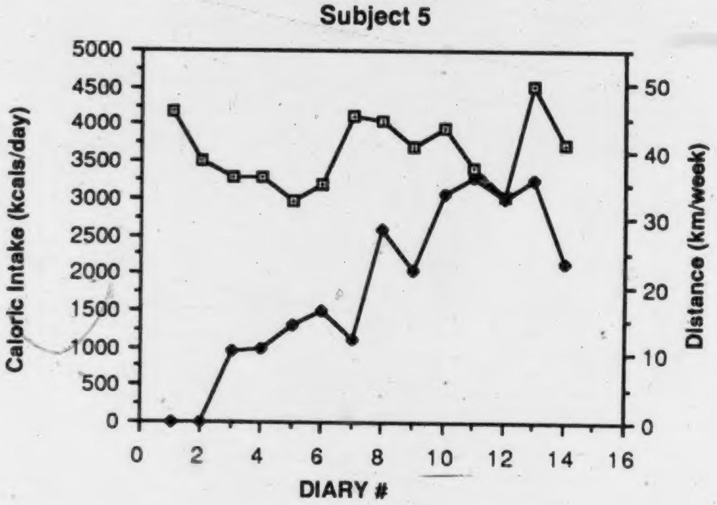


Figure 18e.

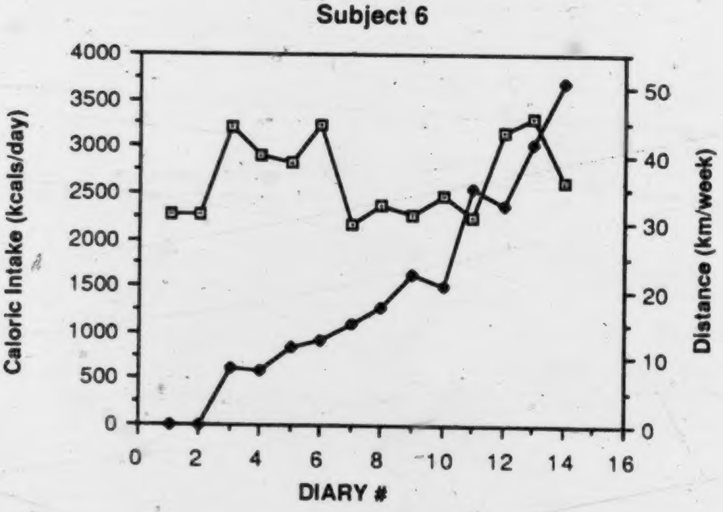


Figure 18f.

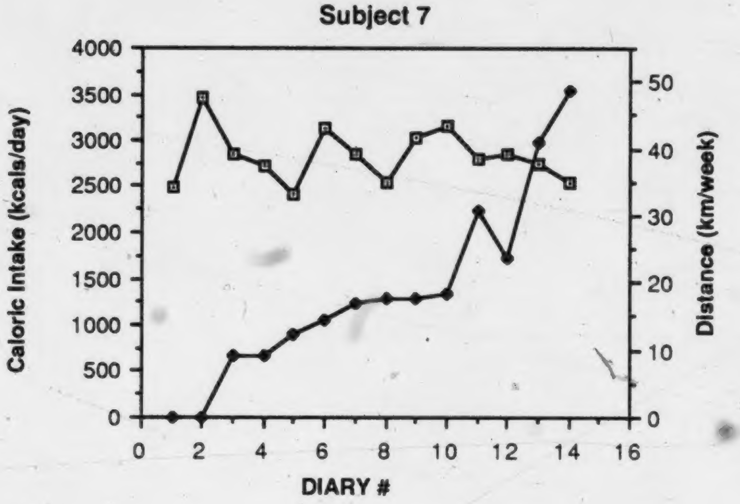


Figure 18g.

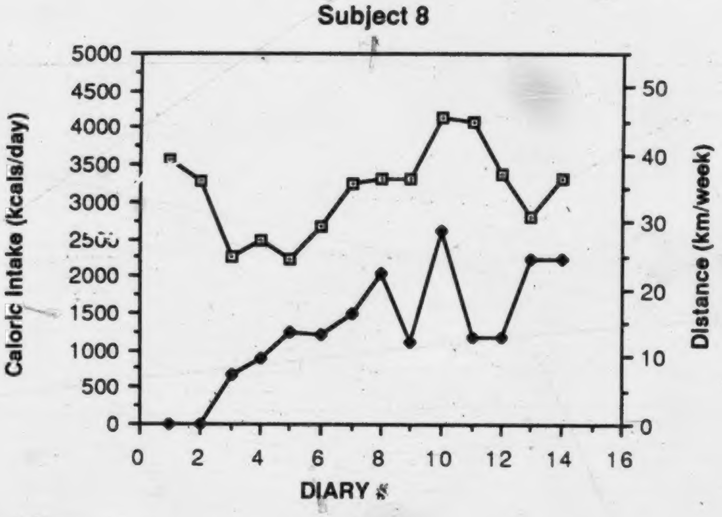


Figure 18h.

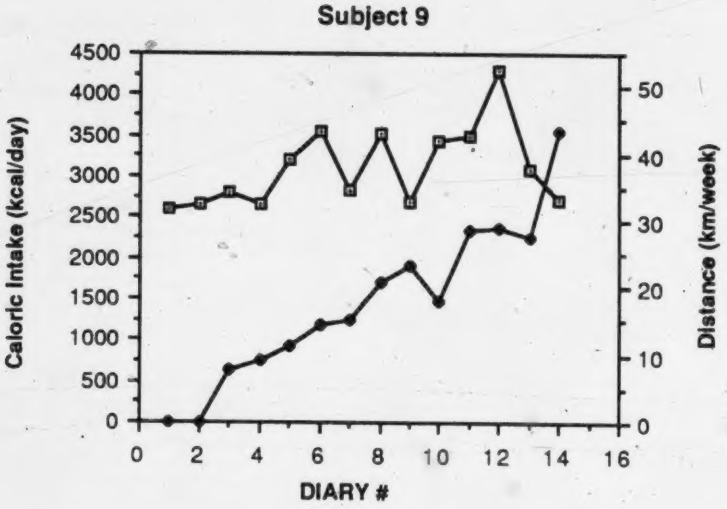


Figure 18i.

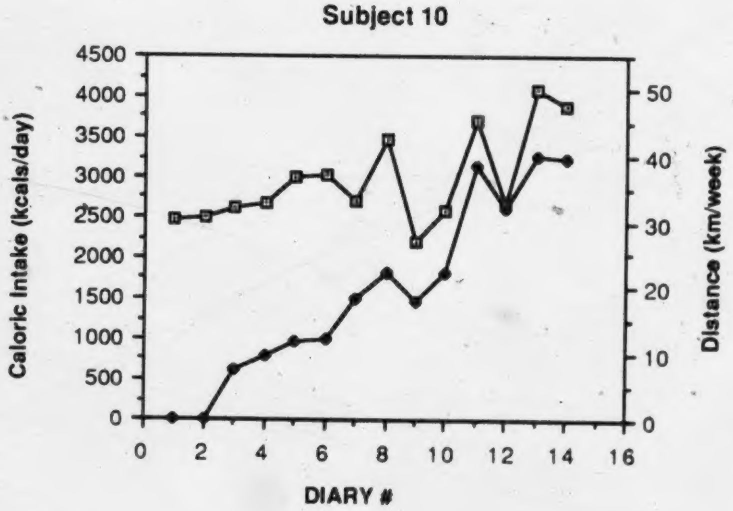


Figure 18j.

Individual training and dietary profiles: TRF

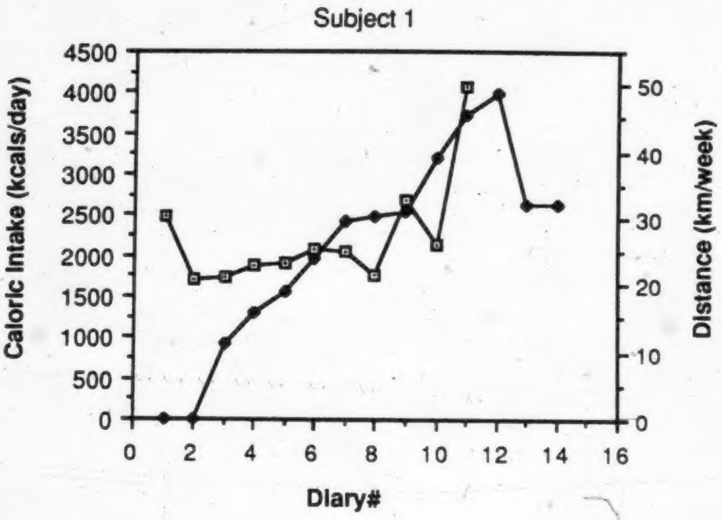


Figure 19a.

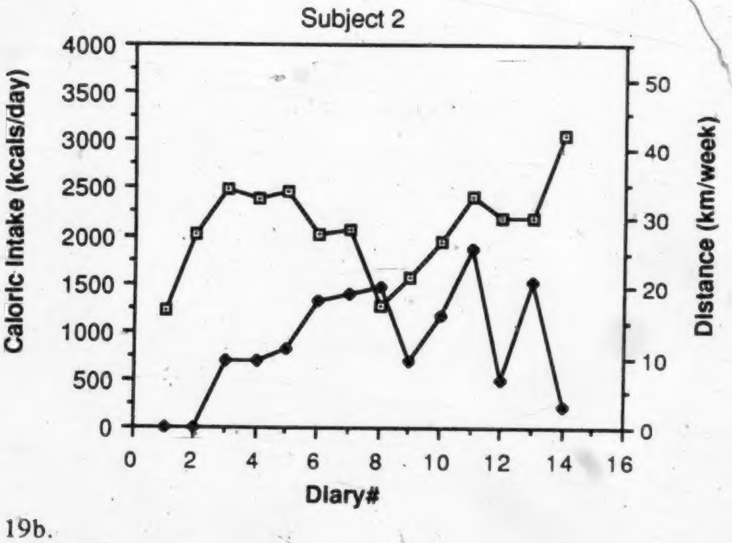


Figure 19b.

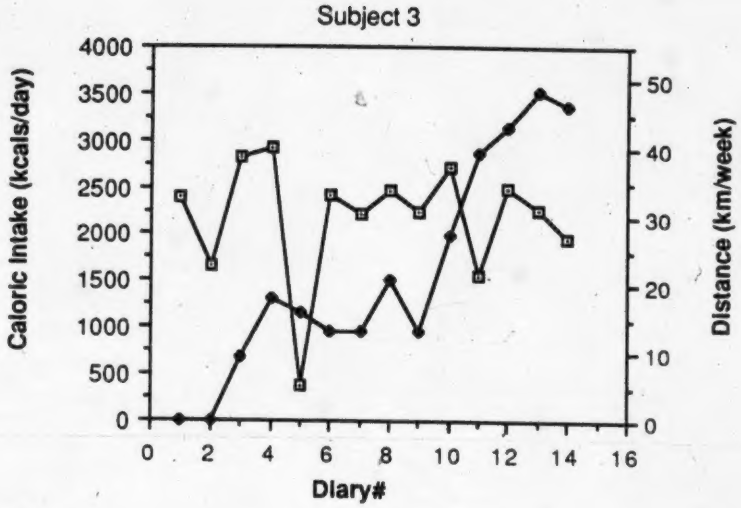


Figure 19c.

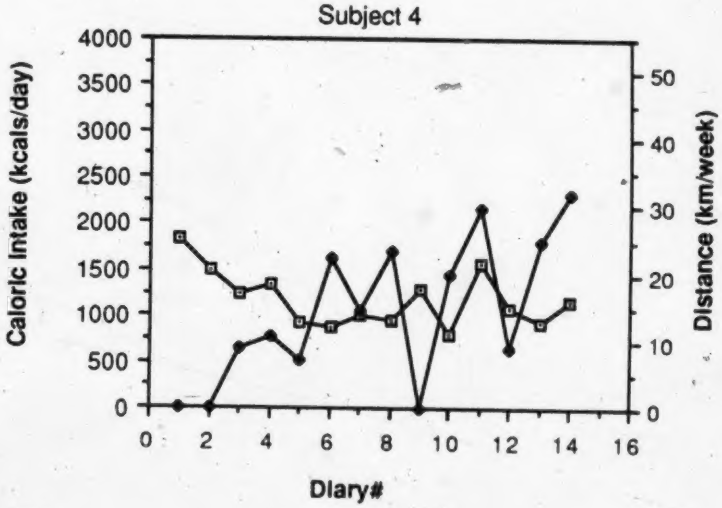


Figure 19d.

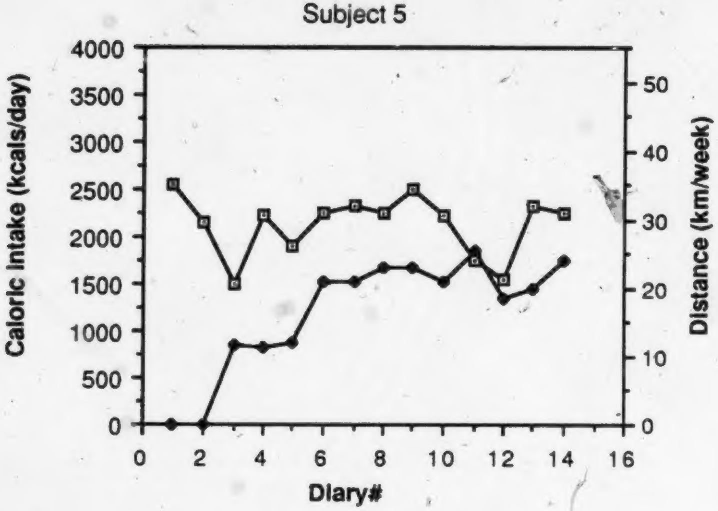


Figure 19e.

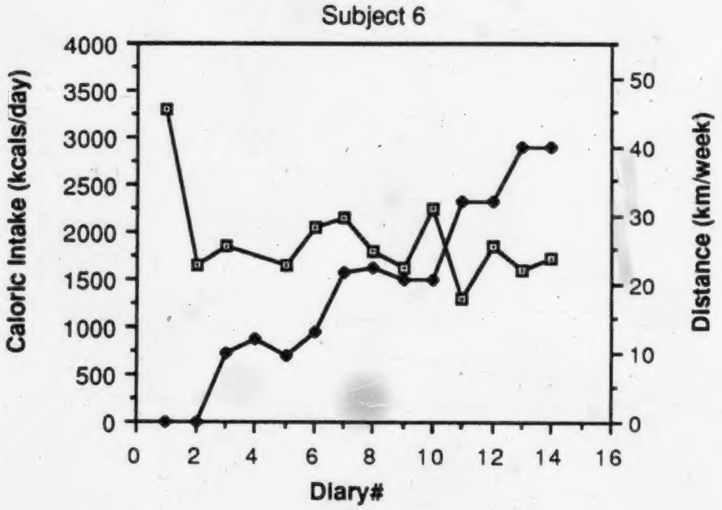


Figure 19f.

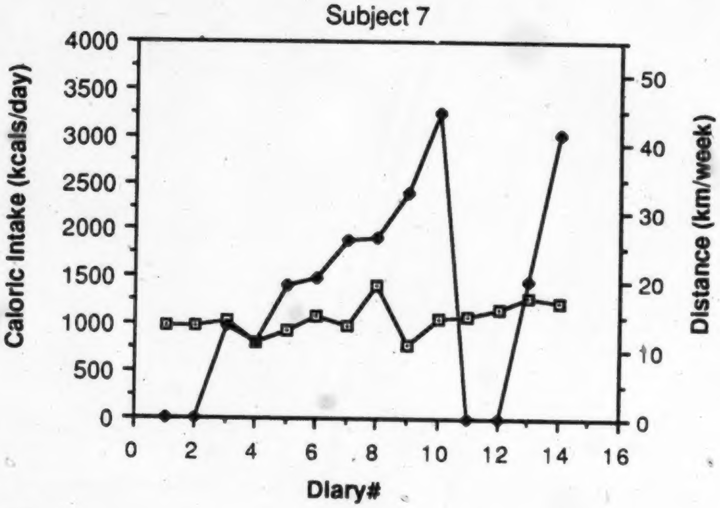


Figure 19g.

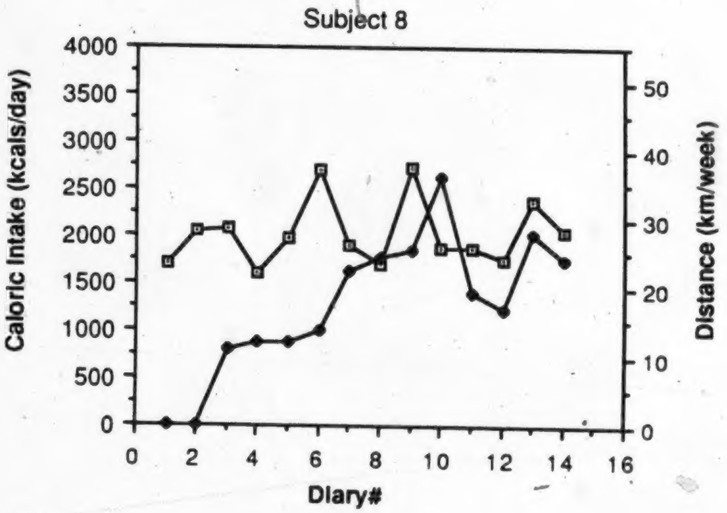


Figure 19h.

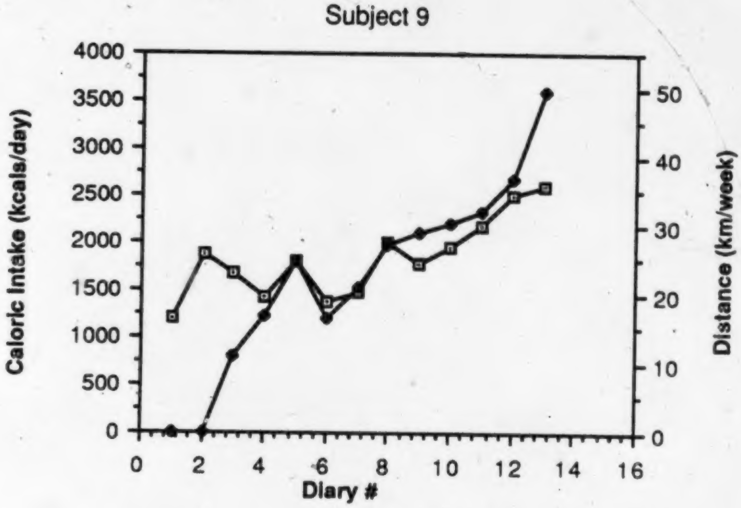

Subject 9
Caloric Intake (kcals/day)
Distance (km/week)
Diary #
4000
3500
3000
2500
2000
1500
1000
500
0
50
40
30
20
10
0
0
2
4
6
8
10
12
14
16

Figure 19i.

Energy Expenditure and Energy Intake (Energy Balance):

Since there were no differences in caloric intake either among groups or with training an analysis of energy balance was completed. It would be expected that an increase in training volume and/or a program of endurance running would necessitate ingestion of a greater number of calories than in a sedentary state. Basal metabolic rate was estimated using the nomogram of Boothby et al. (1936) and Boothby (1956) and the energy cost of exercise was calculated based on a constant for normal daily activity (20% of caloric intake). The specific dynamic action of food was estimated as 6% of total caloric intake for trained and 10% for untrained (Bostick-Reed, 1987). The energy cost of exercise was based on the metabolic cost of running on the treadmill (using the non-protein kilocaloric equivalents for oxygen: 4.79 - 5.01 Kcals/Litre O2). The exercise value was equated with actual training intensity on the road or track. The results are reported below expressed as kilocalories surplus/deficit (see Table 11) and as a ratio from zero to one (see Table 12).

Dietary deficit/surplus in HMR, TR and CON after a six month training period:

Main Effects and Interactions:

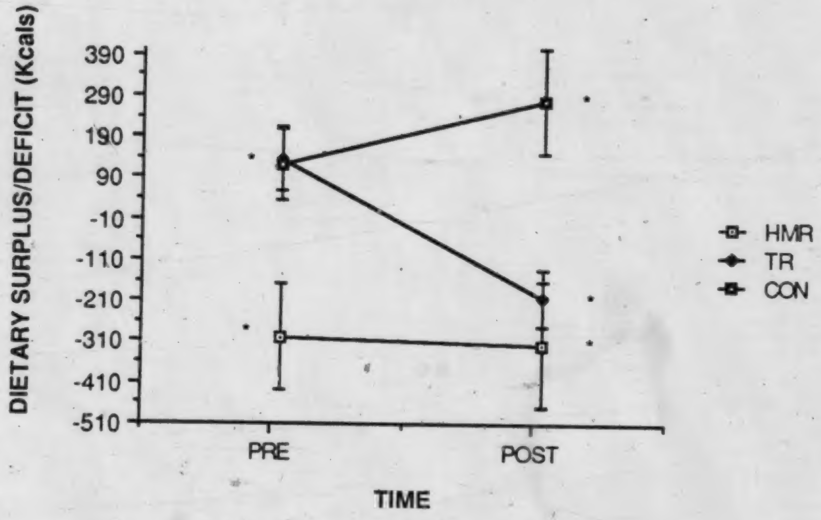
There were highly significant group (p<0.001), group*gender (p<0.01) and group*time (p<0.01) effects for the deficit/surplus measure (estimation). The significant group effect resided in a significantly greater deficit in the HMR group (-313.76 ±165.2 kcals) v TR ([illegible]54.8 ± 54.1 kcals) or CON (+170.90 ± 101.25 kcals). Group*gender multiple comparisons revealed that HMRM (-531.7

± 165.2 kcals) and not HMRF (-43.34 ± 139.1 kcals) had a significantly greater estimated caloric deficit than either the TRM (34.5 ± 60.7 kcals) or CONM (+301.6 ± 117.4 kcals) groups. TRF, HMRF and CONF were not significantly different. Group*time multiple comparisons showed that following training the HMR (-358.36 ± 69.74 kcals) and TR (-215.7 ± 51.4 kcals) groups as a whole exhibited a greater deficit than the CON group whereas prior to training there was no difference. This CON surplus was consistent across groups and time. . Although there was a considerable shift from a caloric surplus in the TR group, (both men and women) to a negative caloric balance, this was not significant due to considerable within group variability.

Mean results for all groups are reported in table 11 and graphically represented in figures 20 to 21.

Table 11. Dietary surplus/Deficit (Kilocalories/day) in HMR, TR and CON.

Group	Pre X sem	Post X sem
HMRM	-579.4	-483.9
	164.9	218.5
HMRF	45.74	-132.43
	118.8	194.6
TRM	210.3	-141.2
	89.6	97.2
TRF	4.81	-318.5
	116.3	116.3
CONM	246.7	356.5
	154.5	161.9
CONF	21.9	198.1
	113.3	176.6

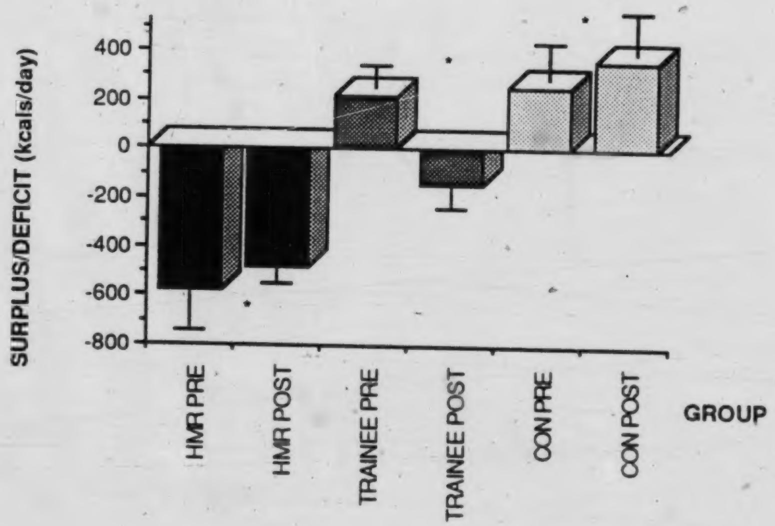


*P<0.05: *Pre HMR sig. greater deficit v TR or CON.*

Post HMR sig. greater deficit than CON but not TR.

No sig. difference between HMR & TR post measure.

Figure 22: Dietary surplus/deficit in HMR,TR and CON subjects before and after training.



* $p<0.05$ Scheffe: *HMR had a significantly greater deficit before, during and after training than TRM and CONM.*

Figure 23: Dietary Surplus/Deficit in HMRM, TRM and CONM subjects.

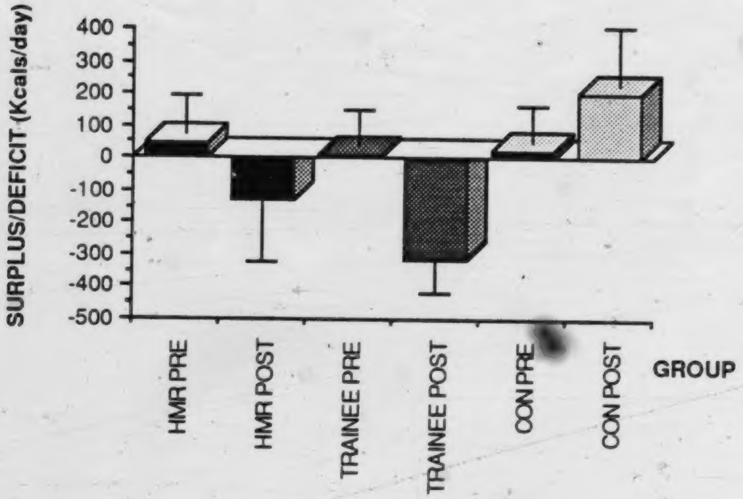


Figure 24: Dietary Surplus/Deficit in HMRF, TRF and CONF subjects.

Dietary Deficit Ratios for HMR, TR and CON before and after training.

Ratios are reported in tabular form (see table 12

Main Effects and effects of training.

Main Effects and effects of training on the ratio are as per absolute caloric surplus/deficit section.

Table 12: Dietary deficit/surplus ratios before and after training.

Group	Pre x sem	Post x sem
HMRM	0.814	0.834
	0.05	0.07
HMRF	1.02	0.95
	0.1	0.08
TRM	1.07	0.95
	0.03	0.03
TRF	0.99	0.86
	0.06	0.04
CONM	1.11	1.15
	0.07	0.07
CONF	1.01	1.12
	0.07	0.1

Comparison of nutrient intake of HMR, TR and CON to the RNI for Canadians.

The low caloric intake of the HMRM group compared to the other exercise and control group was interesting in that there appeared to be no increase in dietary intake to compensate for increased activity (see table 13). HMRM consumed 13% less calories than TRM and 4.5% less calories than CONM. When body weight was factored into the caloric measure there were no differences among the groups; HMRM, TRM and CONM consumed an average of 37.3, 35.8 and 34.7 kcals/kg body weight respectively. The HMRM group exhibited a dietary deficit of -579.37±164.9 kilocalories of energy per day. This compared to a surplus of 210.33± 89.58 Kilocalories in TRM and 246.72 ± 154.52 Kilocalories in CONM at the outset of the investigation. This deficit was consistent and significantly greater than either TRM or CONM ($P<0.05$) within the HMR group over the course of the investigation (post measure = -483.97±218.47 Kilocalories). In contrast to the HMRM group HMRF appeared to consume more absolute calories than either TRF or CONF. HMRF consumed 28.5% more calories than TRF and 30% more than the CONF group. When body weight was factored into the caloric intake measure there was a clear difference in energy consumption. HMRF consumed an average of 42.85 Kcal/kg compared to 33.4 in TRF and 32.9 in the CONF group. It was interesting that HMRF consumed more calories per kilogram body weight than the HMRM group. The diet deficit/surplus values computed for the women subjects also demonstrated a value close to dietary equilibrium. HMRF consumed a surplus of only +45.7 kilocalories whereas TRF consumed +4.81 Kilocalories and CONF, +21.93 Kilocalories.

Since there is little reference data from research studies of this nature with which to compare the current data, caloric intake was compared to the recommended nutrient for Canadians (Dietary standards for Canada, Minister of National Health and Welfare, Ottawa, 1975). The following table compares the nutrient intake of subjects at the initial/pre measurement phase to Canadian dietary standards (see table 13).

Since the Canadian standards are based upon values calculated to be 2 standard deviations above actual required mean caloric consumption then it is evident that the caloric intake of the HMRM group falls short of the requirements. This is particularly evident since this group was training in excess of 70 kms/week and would not appear to compensate for this activity with increased caloric intake. An apparent shortfall in the CONM and CONF groups would not be considered problematic since the RNI for Canadians is set at 2 standard deviations above minimum nutrient requirements for adults.

Table 13. Comparison of caloric intake to Canadian dietary standards (pre investigation measures).

Group	Dietary intake (Kilocals)	Diet req. (Kilocals)	%RNI
HMRM	2440.50	2700 * **	90.0
HMRF	2230.71	2100	106.2
TRM	2750.71	3000	91.7
TRF	2028.00	2100	96.5
CONM	2551.17	3000	85.0
CONF	1780.45	2100	85.0

* caloric intake assumes a normal activity pattern (ie: not inclusive of training regimens).

** values are 2 standard deviations above the mean calculated requirements for each age group.

Table14. A comparison of dietary protein intake in HMR,TR and CON to Canadian dietary standards (Pre experimental measures).

Group	Prot.Intake (gms)	Prot/kg. (gms)	%RNI	%RNI (Athlete)
HMRM	90.0	1.37	160.0	68.0
HMRF	87.4	1.68	213.2	84.0
TRM	111.5	1.45	205.4	
TRF	72.31	1.19	176.3	
CONM	97.2	1.32	173.6	
CONF	69.5	1.28	169.5	

An examination of the table 14 reveals that according to dietary standards for Canadians all groups consumed sufficient total protein. The dietary reference standard of 1 gm/kg body weight was exceeded in all cases. A protein intake of 2.0 gm/kg/body weight has been suggested for athletes. In this case the HMR group may be seen to be consuming only 68% if required intake in HMRM and 84% of required intake in the HMRF group.

An examination of pre measures of total and relative caloric intake and protein thus revealed an apparent dietary deficit in total calories consumed in the HMRM group and a deficit in terms of recommended protein intake in both HMRM and HMRF.

Since the purpose of the investigation was to examine the effects of exercise on caloric intake it is pertinent to examine the effects of exercise on caloric intake and protein consumption over time (ie: during the investigation training period) and relative to the Canadian dietary standards. (table 15)

Table15: Caloric intake in HMR, TR and CON following six months of endurance training: comparison to RNI for Canadians.

Group	Diet Intake	Diet Req.	%RNI
HMRM	2394.0	2700	88.7
HMRF	2127.0	2100	101.3
TRM	2825.0	3000	94.2
TRF	1939.0	2100	92.3
CONM	2695.3	3000	89.8
CONF	1950.5	2100	92.9

There were no changes in dietary protein intake with training or between the groups (see table 16). In relative terms all groups satisifed the daily requirement of at least 1gm protein/kg body weight by a comfortable margin. Again, however, if a value of 2.0 gms/kg is accepted as the requirement for athletes and active individuals then both the HMR.and the TR groups might be considered as dietarily deficient in protein intake.

Table 16: Protein Intake following a six month endurance training program: comparison to Canadian RNI.

Group	Prot.intake (gms)	Prot/kg (gms)	%RNI	%RNI (athlete)
HMRM	90.0	1.37	160.7	69.0
HMRF	79.43	1.54	193.7	76.0
TRM	107.5	1.45	192.0	72.6
TRF	77.5	1.31	189.0	64.5
CONM	106.7	1.45	190.5	
CONF	68.3	1.24	166.5	

The absolute carbohydrate intake of the groups was not significantly different either between groups of over time (see table 17). Since carbohydrates are the main source of fuel of high intensity exercise it was thought that HMRM and HMRF would initially consume more carbohydrates than either of the other groups. This was not the case. However, there was shift towards an increase in carbohydrate consumption in the TR group over the six month period which appeared to have been compensated for by a decrease in dietary fat intake. The increase in carbohydrate intake was not significant.

Table 17: Carbohydrate intake as a percentage of total nutrient intake.

Group	PreCHO(%)	MidCHO(%)	PostCHO(%)
HMRM	61.4	63.3	61.5
HMRF	65.2	65.0	66.7
TRM	60.0	63.1	64.2
TRF	63.3	60.9	60.1
CONM	59.8	58.9	60.3
CONF	59.3	61.2	62.7

There were no differences in dietary fat intake among the groups or over time. There was however, a trend for a decrease in fat consumption as a percentage of total nutrient intake in the TRM group. No other group changed in fat intake (see table 18).

Table 18: Dietary fat intake as a percentage of total nutrient intake.

Group	PreFAT(%)	MidFAT(%)	PostFAT(%)
HMRM	19.3	17.3	19.4
HMRF	16.6	16.2	15.1
TRM	19.4	18.2	16.7
TRF	18.4	19.3	18.9
CONM	20.8	21.8	19.4
CONF	21.1	20.0	19.8

Effects of a six month training program on Eating Attitudes and Eating Disorder Inventory Scores.

Main Effects: Eating Attitudes Test.

There were significant group (p<0.01) and gender (p<0.01) main effects for the EAT but no other interaction was significant. A significant group effect was accounted for by a difference between TR (9.3 ± 1.6 points) and CON (3.5 ±0.85 points) (p<0.05 Scheffe) (see table 19). HMR (5.91 ± 1.25 points) did not differ from TR and CON by group (see figure 25). Post hoc Scheffe multiple comparisons revealed that the group difference between TR and CON was accounted for by a significantly higher EAT score in TRF (13.9 ± 2.5) than in CONF in the pre measure (4.16 ± 1.1)(p<0.05 Scheffe) (see figure 27). TRM (5.36 ± 1.5) also scored significantly lower on the EAT than TRF (p<0.05). Post hoc multiple comparison revealed a significant pre measure difference among men and women but not a post measure difference (p<0.05). The group*gender*time interaction was not significant.

The Eating Disorder Inventory:

There were significant group (p<0.01) and gender (0.001) main effects for the EDI scores among the three groups. A significant group effect was accounted for by significantly higher EDI scores in the TR (19.38 ± 2.7) than HMR(7.0 ± 1.06) group (see figure 26). Post hoc multiple comparisons revealed that the group difference was accounted for by significantly higher EDI scores in TRF (28.54 ± 3.7) than HMRF (7.75 ± 1.91) (p<0.05 Scheffe) (see figure 28). There was also a within group difference between TRF (28.54 ± 3.7) and

TRM (11.54 ± 2.55) on EDI scores ($p<0.05$ Scheffe). There was a significant group*time interaction effect. Multiple comparisons over time showed that there was a before and after difference in EDI scores in men and women ($P<0.05$).

Effects of training on EAT and EDI scores in HMR,TR and CON.

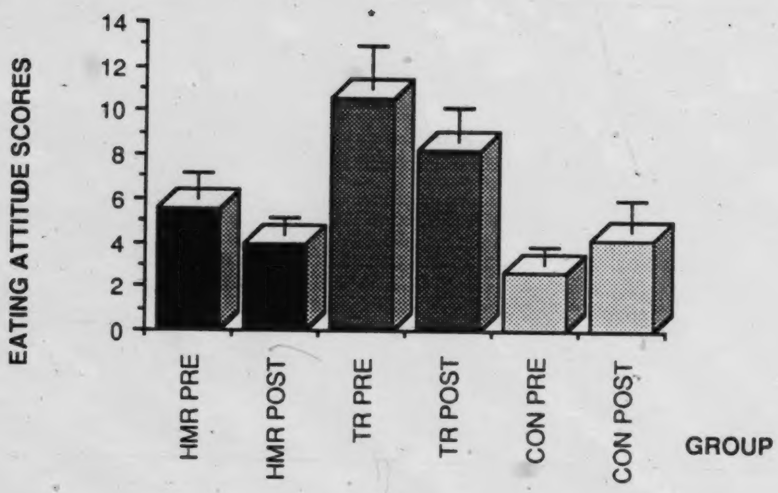
There was no effect of training on either the EDI or EAT scores in the HMR group (Table 19). Nor was there any within gender difference in scores over time in this group. Neither HMRM or HMRF scored any differently on either score than TR or CON groups except on the EDI on which the HMRF group scored significantly lower than the TRF group ($p<0.05$ Scheffe). Although the TRF scores were much higher than the HMRF scores on both the EAT and the EDI there were no significant differences when Scheffe multiple comparisons were calculated.

Six months of endurance running training did nothing to alter the eating attitudes or the EDI scores of the TR group (figures 25 and 26). However, there was a noticeable trend for much higher scores both before and after training in the TRF group. On the EAT, TRF scored significantly higher scores overall than CONF and TRM ($p<0.05$ Scheffe). TRF also scored significantly higher than the HMRF and TRM groups on the EDI ($p<0.05$) (figures 27 and 28).

There were no changes in the EAT or EDI scores in the CON group over time. Like the TRF group, the CONF group tended to score higher EDI scores than the HMRF group although the differences were not significant. TRF scored much higher scores on the EAT than the CONF group ($p<0.05$) but not on the EDI. As a group the TR group scored higher scores on the EAT than the CON group on the pre measure but not the post measure ($p<0.05$ Scheffe).

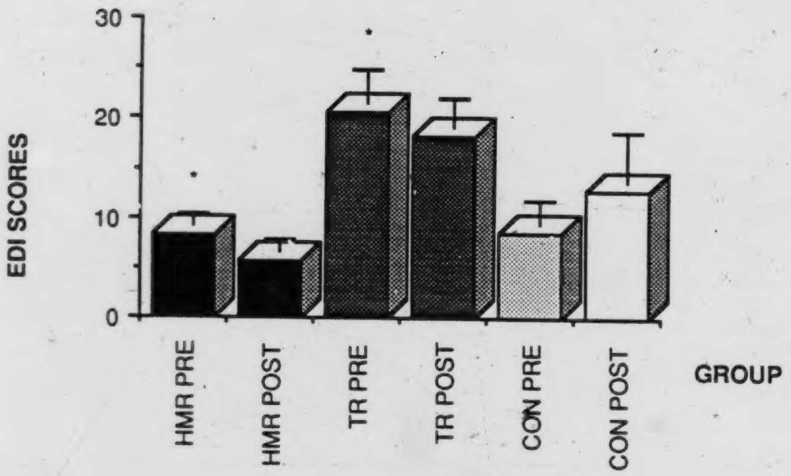
Table 19. EAT and EDI scores in HMR, TR & CON before and after training.

GROUP	GENDER	EAT		EDI	
		PRE (x/sem)	POST (x/sem)	PRE (x/sem)	POST (x/sem)
HMR	M	4.67	2.56	8.00	4.20
		1.5	0.6	0.8	0.7
	F	7.00	8.30	8.50	7.00
		1.8	1.48	2.23	1.65
TR	M	5.93	4.78	12.21	10.80
		1.6	1.5	3.0	2.2
	F	15.68	12.16	30.3	26.75
		3.2	2.4	4.9	4.8
CON	M	1.60	2.20	4.20	6.00
		0.8	1.4	1.4	2.1
	F	3.20	5.10	10.36	15.81
		0.9	1.9	3.4	7.2



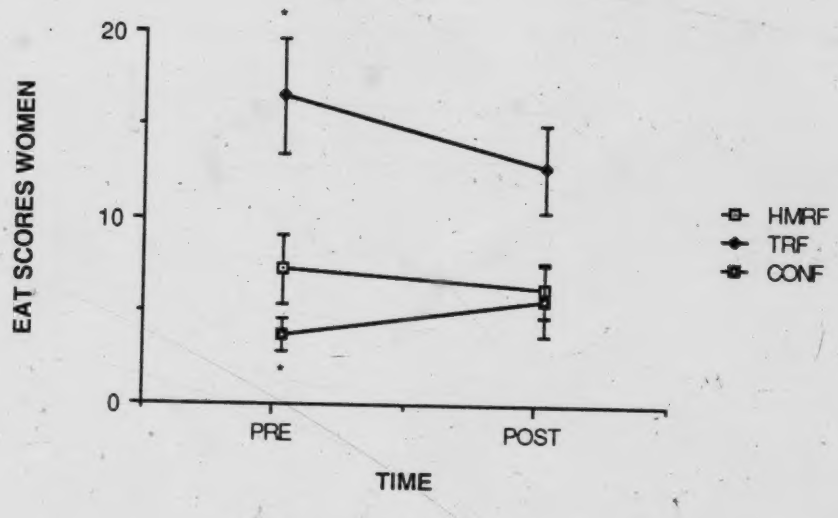
*P<0.05 (Scheffe): *TR scored significantly higher EAT scores than the control group before and after training. Differences were accounted for by significantly higher scores in the TRF v CONF groups.*

Figure 25: EAT Scores by group before and after training.



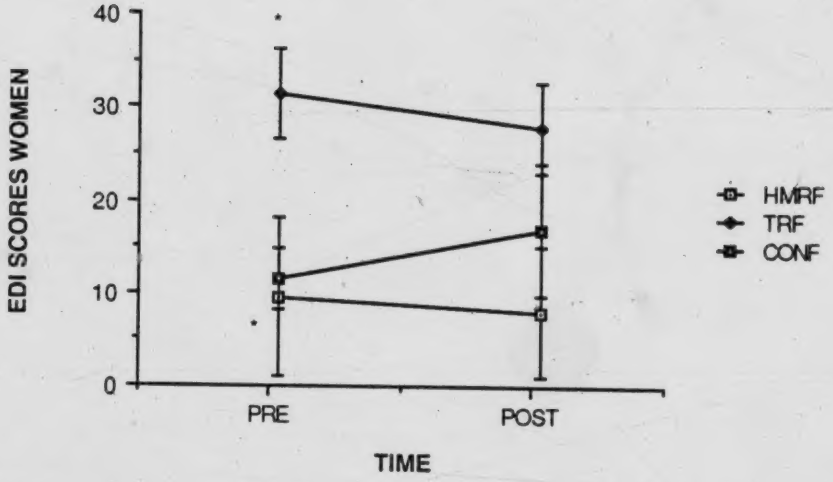
*P<0.05: *TR scored significantly higher EDI scores than the HMR group but not CON. Differences between HMR and TR were accounted for by significantly higher EDI scores in TRF v HMRF.*

Figure 26: EDI scores by group before and after training.



*P<0.05 (Scheffe): *TRF scored significantly higher EAT scores than CONF.*

Figure 27: EAT scores: group*gender (women)



*P<0.05 (Scheffe): *TRF scored significantly higher EDI scores than the HMRF group.*

Figure 28: EDI scores: group*gender (women)

Result section 5. The effects of six months of endurance training on serum Testosterone, SHBG capacity, Free Androgen Index, Prolactin, Cortisol, LH, FSH and Pulsatile LH release in HMR and TRM subjects.

Main Effects:

Serum total testosterone was significantly different in HMR (594.2 ± 58.1 ng/dl) v TRM (885.4 ± 59.1 ng/dl) at the onset of the investigation ($p<0.05$ Scheffe). After training there was no significant difference between HMR (586.5 ± 73.1 ng/dl) and TRM (684.1 ± 58.3 ng/dl). TRM pre testosterone levels were 23% greater than pre HMR testosterone levels. There was a significant reduction in serum testosterone from pre to post training period in the TR group (885.4 ± 59.1 to 684.1 ± 58.3 ng/dl) ($p<0.05$ Scheffe) (see figure 29). The drop in testosterone represented a 22% decrease in circulating levels. There were no significant group or time main effects in sex hormone binding capacity (SHBG) (see figure 30).

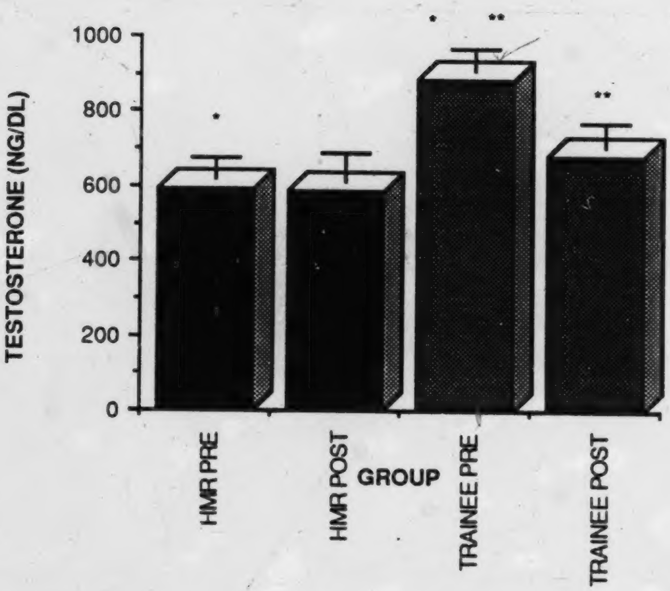
Although the Free Androgen Index was considerably greater in the TR v HMR group there were no group or group*time differences in the Free Androgen Index (T/SHBG ratio) (see figure 31). Post hoc Scheffe comparison revealed that HMR post FSH levels were significantly higher than either TRM pre or post values ($p<0.05$, Scheffe) (see figure 32).

There was a significant reduction in cortisol levels in TRM ($p<0.05$) although the alterations in pre and post levels were not physiologically significant ie: were within the normal range (see figure 33).

LH levels did not differ between groups or over time in either group (see figures 34 &35). There were no group differences in prolactin levels although there was a significant reduction in prolactin in the TRM group (11.6 ± 0.8 ng/ml to 8.9 ± 0.6 ng/ml)(see figure 36). Mean values for hormonal measures are reported in table 20.

Table 20. Hormonal changes during a six month training program.

GROUP	TIME	TEST ng/dl	SHBG mmol/l	FAI	FSH (mIU/ml)	LH	F ng/ml	PRL ng/ml
HMR	PRE	594.2	37.9	64.5	12.9	11.4	6.0	10.9
		58.1	5.1	10.2	1.9	0.9	0.1	1.58
	POST	586.5	36.6	58.7	15.2	_	5.7	8.7
		73.1	2.6	8.7	2.6	_	0.5	1.5
TRM	PRE	885.4	31.9	116.6	7.9	10.2	7.2	11.6
		59.1	3.6	15.8	0.8	0.3	0.5	0.8
	POST	684.1	31.1	97.6	7.5	9.8	5.4	8.9
		58.3	3.5	16.7	0.9	0.7	0.4	0.6

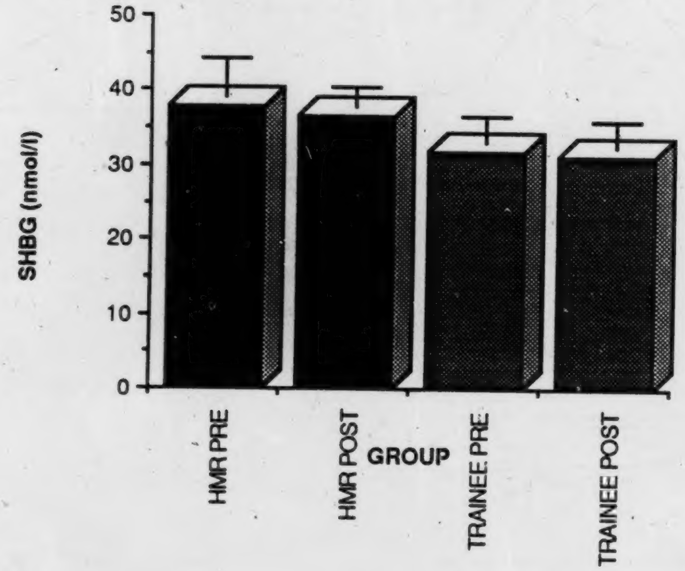


* ** all $P<0.05$ (Scheffe):

** HMR pre T levels were significantly lower than pre T of TR group but not post training TR levels. HMR T levels did not change as a function of training season.*

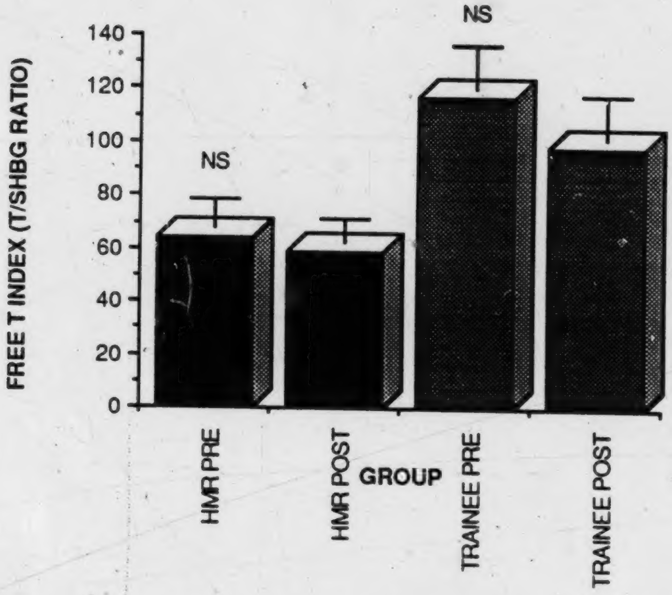
*** Training resulted in a significant decrease in total T in the TR group.*

Figure 29. Effects of training on total Testosterone in HMR & TR subjects.



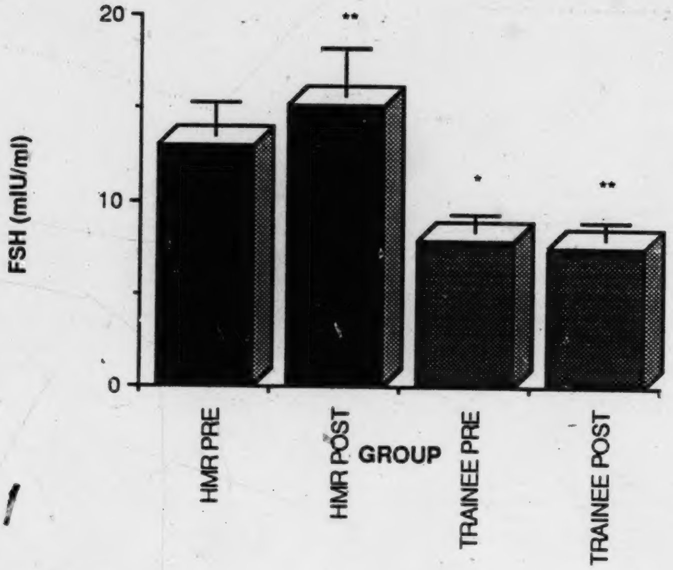
NS: There was no effect of time or training on the SHBG capacity of either HMR or TR subjects.

Figure 30. Effects of training on SHBG capacity in HMR and TR subjects.



NS: *Although there was a considerable difference between HMR and TR in the FAI before and after training these differences were not significant. A decreased FAI with training in the TR group was also not significant.*

Figure 31. Effects of training on the Free Androgen Index in HMR and TR subjects.

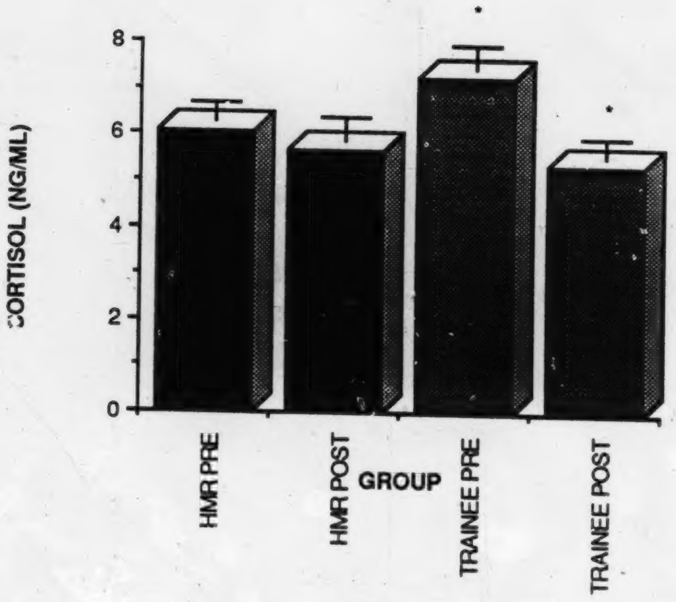


* ** all P<0.05 (Scheffe):

** Pre TR FSH levels were significantly lower than pre HMR levels.*

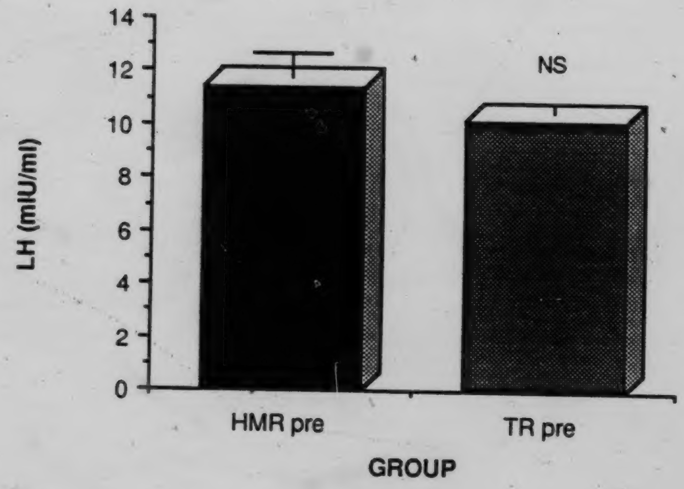
*** Post FSH levels in HMR were significantly higher in the HMR group.*

Figure 32. Effects of training on FSH levels in HMR and TR subjects.



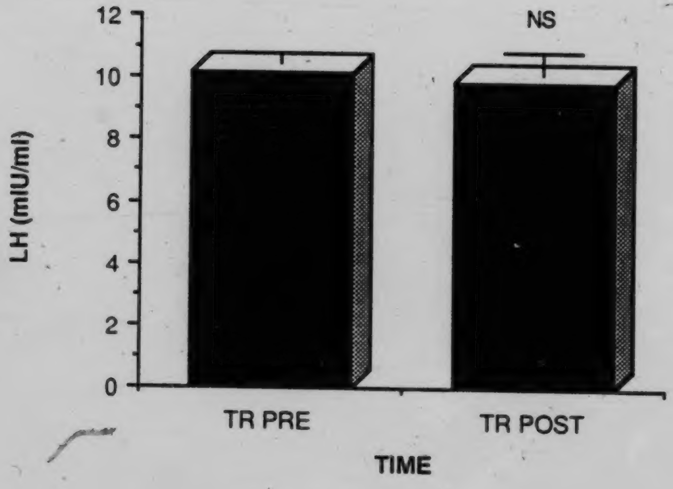
* $P<0.05$ (Scheffe): *There was a significant drop in baseline cortisol levels after six months of endurance training in the TR group. There was no change in the HMR group.*

Figure 33. Effects of training on Cortisol in HMR and TR subjects.



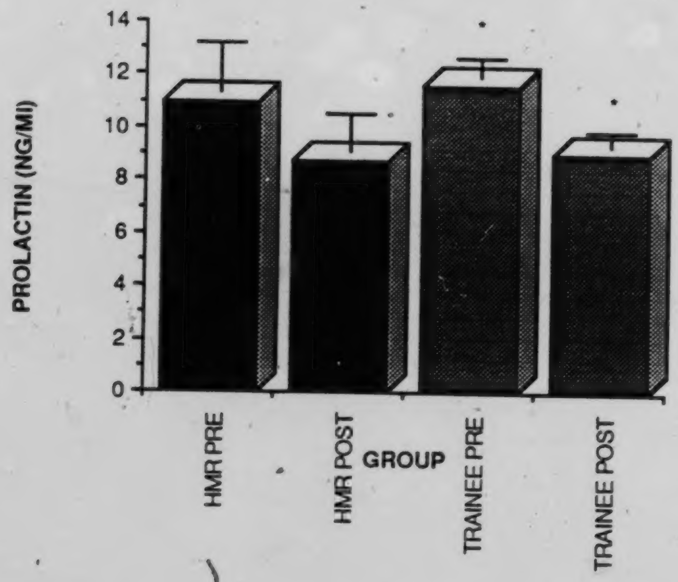
NS: *Baseline LH levels were not significantly different between TR and HMR.*

Figure 34. Baseline LH levels in HMR and TR.



NS: *Training had no effect on baseline mean LH levels in the TR group.*

Figure 35. Effects of training on baseline LH levels in TR subjects.



* $P<0.05$ (Scheffe): *Training resulted in a significant drop in Prolactin levels in the TR group.*

Figure 36. Effects of training on Prolactin in HMR and TR subjects.

The Effects of six months of endurance running training on LH pulsatile release in HMR and TR subjects.

Comparison of LH pulse parameters between HMRM and TRM.

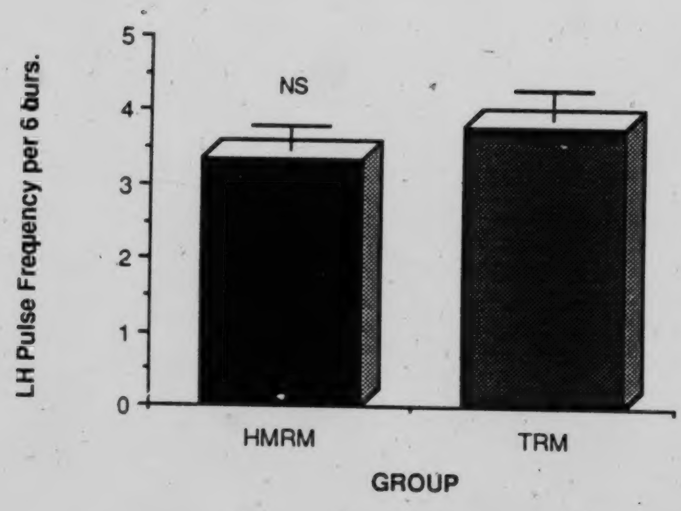
The effects of training in LH pulsatile parameters were measured in the HMRM and TRM groups only. Differences between the Pre measures between HMRM and TRM were assessed via a one way ANOVA. Differences in pulse parameters between pre and post LH pulse parameters in the TRM group were assessed via a one group T test for dependent measures (2 tailed).

There was a small but insignificant difference in the number of LH pulses (peaks) between HMRM and TRM. No other LH pulse parameters were different (Table 21, figure 37).

Table 21. Summary of statistics: HMR V TRM: LH pulse Parameters.

Variable	HMRM	TRM	Prob.
Mean LH	11.42	10.20	0.282
	0.93	0.30	
Amplitude	3.23	4.41	0.292
	0.38	1.17	
Nadir	10.04	8.84	0.297
	0.94	0.36	
Pulse Area	130.1	133.8	0.926
	30.2	22.2	
Pulse Interval	132.1	102.2	0.343
	25.3	11.6	
Pulses	3.33	3.80	0.379 NS
	0.33	0.37	
Area Under LH Curve.	NA	NA	NA

NS: *There were no significant differences among any of the pulse pattern variables between HMR and TR.*



NS: *There was a small but insignificant difference in the number of LH pulses per 6 hours between TR and HMR.*

Figure 37. Mean LH Pulse Frequency in HMRM and TRM.

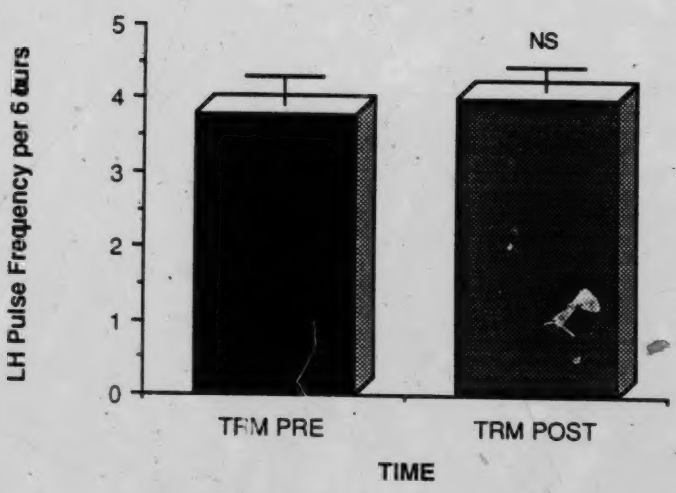
The effects of six months of endurance training on LH pulsatile release in TRM.

Table 22. Summary of T Test Results: LH Pulse parameters before and after training in TRM.

Variable	TRM Pre	TRM Post	Probability.
Mean LH	10.2	9.86	0.652
	0.34	0.69	
Pulse Freq.	3.8	4.0	0.561
	0.37	0.32	
Nadir	8.84	8.41	0.462
	0.36	0.53	
Pulse Area	133.8	139.3	0.876
	22.2	33.1	
Pulse Amplitude	4.41	4.15	0.793
	1.17	0.93	
Pulse Interval	102.2	78.75	0.0078 *
	11.7	4.74	
Area Under LH Curve	NA	NA	NA

* *Only mean pulse interval was significantly different* ($P<0.01$).

There were no significant effects of training on the pulse pattern profile of the TRM group except for a significant drop in the mean pulse interval from pre to post investigation measurement ($P<0.01$). A small decrement in baseline LH levels was not significant (Table 22, figure 38).



NS: *Training did not result in a change in LH pulse frequency in the TR group.*

Figure 38. Mean LH Pulse Frequency in TRM before and after training.

Results Section 6: Correlations among variables.

To examine the relationship among the variables measured, correlation coefficients and matrices were computed. A factor analysis was also computed to determine the relationship between endocrine and selected nutritional factors.

Correlation Matrices:

Correlation matrices were computed for all nutritional variables versus pre and post testosterone levels by individual group and pooled group data. Correlation matrices are located in the appendices.

Simple Correlation Coefficients: The relationship of Testosterone levels to pre and post variables:

To examine the relationship of selected nutritional variables to pre and post training testosterone levels, correlation coefficients were computed for caloric intake, caloric intake per kg body weight, caloric intake per kg LBM, CHO, fat, protein (absolute (gms) and relative to body weight (gms/kg)), caloric deficit (absolute and per kg body weight). Correlations were computed on a pre and post training basis for HMRM only, TRM only and for HMRM and TRM as a group. Results are published below in tables 23, 24 and 25

Table 23. Correlation coefficients for HMR: Pre and Post Investigation: v pre and post Testosterone levels.

Variable	Pre Coeff.	sig	Post Coeff.	sig
Caloric Intake	0.764	0.01	0.570	ns
Cal/kg/wt	0.782	0.01	0.503	ns
Cal/kg/LBM	0.817	0.01	0.600	0.05
Deficit	0.367	ns	0.510	ns
Deficit/kg/wt	0.374	ns	0.531	ns
Protein	0.726	0.01	0.697	ns
Protein/kg/wt	0.786	0.01	0.640	0.05
Protein.kg/lbm	0.819	0.01	0.747	0.01
Fat	0.857	0.01	0.615	0.05
Fat/kg/wt	0.844	0.01	0.547	ns
CHO	0.442	ns	0.066	ns
CHO/kg/wt	0.520	ns	0.040	ns

Table 24: Correlations: pre and Post training: TRM group.
Selected variables v Testosterone.

Variable	PreCoeff.	sig	PostCoeff.	sig
Caloric Intake	-0.532	0.05	0.352	ns
Cal/kg/wt	-0.687	0.01	0.326	ns
Cal/kg/LBM	-0.733	0.01	0.197	ns
Deficit	-0.494	0.1	0.302	ns
Deficit/kg/wt	-0.521	0.05	0.322	ns
Protein	-0.517	0.05	0.130	ns
Protein/kg/wt	-0.587	0.05	0.082	ns
Prot/kg/lbm	-0.329	ns	0.197	ns
Fat	-0.162	-0.162	0.282	ns
Fat/kg/wt	-0.171	ns	0.255	ns
CHO	-0.437	ns	0.024	ns
CHO/kg/wt	-0.415	ns	0.051	ns

Table 25: Correlation Coefficients: Grouped data (HMR & TR) pre and post training: v testosterone.

Variable	Pre Coeff.	sig	Post Coeff.	sig
Caloric Intake	0.220	ns	0.498	0.02
Cal/kg/wt	0.033	ns	0.415	0.05
Cal/kg/LBM	0.062	ns	0.422	0.05
Deficit	0.359	0.1	0.445	0.05
Deficit/kg/wt	0.380	0.1	0.464	0.02
Protein	0.224	ns	0.463	0.02
Protein/kg/wt	0.175	ns	0.363	0.1
Prot/kg/lbm	0.068	ns	0.361	0.1
Fat	0.314	ns	0.440	0.05
Fat/kg/wt	0.175	ns	0.357	0.1
CHO	0.033	ns	0.134	ns
CHO/kg/wt	0.073	ns	0.091	ns

Description of Correlations:

Training Group:

The most striking trend observed from the calculation of correlation coefficients for this group was a shift from negative correlations of nutritional variables with pre testosterone levels to positive correlations following six months of endurance training. The shift was not, however, significant (see table 24).

HMR group:

There was an extremely strong relationship between pre nutritional variables and pre testosterone levels. This relationship held for the post measurement (see Table 23).

Grouped data:

The most noticeable trend was for a shift towards a stronger correlation of post testosterone to selected nutritional variables. Although these correlations were not significant this does suggest a positive relationship between elements of nutrient intake and androgen levels in men (see Table 25).

Stepwise Regression Analysis:

To further examine the relationship of nutritional variables, absolute and relative measures of caloric intake and dietary deficit with pre and post training testosterone levels data was entered into a stepwise regression analysis. The regression analysis for the absolute and relative variables did not select any significant predictors of pre testosterone in the HMR and TR pooled data. However, a stepwise regression analysis revealed two significant predictors of post testosterone levels. Absolute caloric intake was the only significant variable in the first analysis based on absolute values of nutrients for post data ($p<0.05$) and in the relative analysis the caloric deficit per kg body weight was the most significant predictor of post testosterone ($p<0.05$)

Factor Analysis:

Factor analysis were performed on the nutrient intake variables and testosterone as further indices of the relationship of caloric intake and dietary composition to circulating sex steroid levels.

Factor Analysis: Pre & Post data (HMR & TR).

Principle component analysis and orthotran/varimax rotations produced 3 factors in first analysis.

The orthoganal transformation solution-varimax rotation revealed that the nutrient variables load on factor 1, fat and fat per kg/wt loads on factor 2 and dietary deficit and pre testosterone levels are correlated with factor 3 ie: apparently unrelated to factor 1: the nutrient factor (see Table 26 below). The post measure factor analysis isolates two factors only (see Tables 27 and 28). Absolute and relative fat intake was highly correlated with the nutrient intake factor and total testosterone and dietary deficit were also related to this first factor. The oblique solution to the analysis revealed a stronger relationship of testosterone to factor 1 whereas the importance of carbohydrate intake diminishes significantly.

Table 26. factor Analysis: Pre measures: Orthoganal transformation-varimax:

Variable	Pre Measure		
	Factor1	Factor2	Factor3
Cals/kg	0.819	0.399	0.163
Cals/lbm	0.795	0.412	0.208
Fat/kg	0.319	0.911	0.059
Prot/kg	0.663	0.485	0.220
CHO/kg	0.935	-0.008	0.032
Fat/lbm	0.296	0.914	0.091
Prot/lbm	0.647	0.496	0.262
CHO/lbm	0.937	-0.003	0.079
Def/kg	0.340	-0.016	0.814
PreT	-0.230	0.201	0.822

Table 27: Factor Analysis: Post measures: Orthoganal solution-varimax.

Variable	Post Measure	
	Factor1	Factor2
Cals/kg	0.974	-0.044
Cals/lbm	0.981	-0.021
Fat/kg	0.852	0.221
Prot/kg	0.803	0.283
CHO/kg	0.731	-0.652
Fat/lbm	0.843	0.240
Prot/lbm	0.777	0.310
CHO/lbm	0.739	-0.636
Def/kg	0.898	-0.083
PostT	0.499	0.503

Table 28: Factor analysis: Post measures: Oblique Solution: Primary Pattern.

Variable	Post Measure	
	Factor1	Factor2
Cals/kg	0.636	0.565
Cals/lbm	0.660	0.547
Fat/kg	0.763	0.259
Prot/kg	0.779	0.177
CHO/kg	-0.015	0.984
Fat/lbm	0.772	0.237
Prot/lbm	0.783	0.138
CHO/lbm	0.003	0.975
Def/kg	0.553	0.559
PostT	0.711	-0.214

Chapter 4. A Discussion of the Results:

The discussion of results is arranged such that each of the hypotheses proposed at the beginning of the investigation is examined in the light of current research and findings of this investigation.

Part 1: The effects of exercise on measures of aerobic capacity and percent body fat.

An endurance training program was utilized to examine the effects of training on VO2 max., percent body fat and lean body mass. Furthermore, measures of aerobic capacity, body weight, percent fat and lean body mass were utilized as control measures to demonstrate that a training effect had occurred. Changes in VO2 mass were consistent with those reported in the literature for healthy men and women (see review). Both TRM and TRF exhibited a significant increase in VO2 max. TRM exhibited a 22% increase in VO2 max. and TRF showed a 27% increase in VO2 max. Both groups also achieved a significant increase in training distance over the six month investigation. TRM increased training volume to 39.5 km/week and TRF, to 35.3 km/week. These distances represent training increases from a baseline of no training. Surprisingly only TRM demonstrated a significant decrease in body weight although TR as a group exhibited a significant decrease in percent body fat. This is particularly interesting since the alterations in dietary patterns observed in TRM were not observed in the TRF group (see section 2 of discussion).

Part 2: An examination of the results as they support a model of Activity Anorexia:

The hypotheses proposed were:

1) Six months of endurance running training will result in significant reductions in caloric intake in previously sedentary healthy men and women. High mileage men and women runners will consume significantly less calories than non-exercisers despite their activity level.
2) Dietary intake and dietary behaviour in runners and trainees will support a model of human activity anorexia.

An examination of the nutritional data revealed several interesting findings.

1. High mileage male runners did not appear to consume adequate calories to compensate for normal daily activity and energy expenditure due to training. This was confirmed by a significant calculated dietary deficit in the HMRM group.

2. Training failed to elicit a compensatory increase in caloric intake in the training group for increased energy expenditure.

3. Both the TR and HMR groups consumed no more calories than a group of sedentary control subjects.

4. Although heavier than the CON group the TR group exhibited a negative calorie balance at the end of the investigation. The TR group therefore

appeared to maintain higher body weight on relatively less caloric intake than non-runners.

5. There was a significant quadratic and quartic trend in dietary intake in 5 of the TRM group (responders) although this was not a universal effect. Analysis revealed that the rate of increase in daily running was greater in the responders (initial decrease in caloric intake) and non-responders. This supported earlier findings in animals (Tokuyama et al. 1982).

Anorectic Effect of Exercise:

Animal research has suggested that exercise results in an initial decrease in caloric intake which eventually recovers to above baseline (Tokuyama et al. 1982). Alterations in caloric intake in the rat are associated with daily rate of activity increase (Epling and Pierce, 1984) and alterations in food schedules may result in increased activity (see Epling and Pierce, 1988). Epling and Pierce (1988) have proposed a model of Activity Anorexia which is essentially based on:

1) Opportunity to exercise.
2) Food schedule and/or deprivation.
3) Rate of change of daily activity.

If the animal model of activity anorexia applies to humans then it would be expected that training would result in a decrease in energy consumption and that exercise would increase. The greater the rate of increase in running the greater the appetite suppression effect. Alterations in dietary behaviour, particularly the frequency of meal consumption would be expected to further increase running.

There was no apparent increase or decrease in caloric intake in the TR group with training. An examination of individual dietary profiles of the TRM group, revealed a significant quadratic ($p<0.04$) and quartic ($p<0.02$) trend in dietary behaviour which appeared to be related to exercise intensity; more specifically to the rate of change in running during the first half of the investigation. There was a significant difference in rate of change of running in 5 responders vs 5 non-responders ($p<0.01$). The responders exhibited a 60% greater rate of change in daily running than the non-responder group.

This effect was not observed in the TRF group. This is most likely explained by the fact that TRF did not achieve the same training distances as the TRM and did not exhibit the same rate of change in running as the men. It was interesting that the TRF group tended to follow the guide-lines for training set out by the investigators at the beginning of the training program whereas the TRM often increased training distances above the prescribed rate.

The significant trend in caloric intake in the responder TRM group is consistent with animal investigations (Tokuyama et al. 1982).

The effect of food deprivation and or schedule is unclear in the present investigation. What was clear, was that many HMR and a sub-group of the TR group consumed only one or two meals per day. This was surprising since the degree of energy expenditure would appear to necessitate the ingestion of more calories. The runners therefore appeared to eat too little food and too few meals.

Alterations in Basal Metabolism, Food Efficiency and Dietary Deficit:

The low caloric intake of the HMRM group compared to the other exercise and control group prior to the investigation was interesting in that there appeared to be no increase in dietary intake to compensate for increased activity. An initial measure of the caloric intake of the male groups revealed that HMRM consumed 13% less calories than TRM and 4.5% less calories than CONM. However, the difference was not significant. When body weight was factored into the caloric measure there were no differences among the groups; HMRM, TRM and CONM consumed an average of 37.3, 35.8 and 34.7 kcals/kg body weight respectively. Since there did not appear to be an increase in caloric intake to compensate for physical activity, a diet deficit/surplus value was computed. This was based on an estimate of basal metabolic rate (Boothby, Berkson and Dunn, 1936; Boothby, 1956, in Bostick-Reed, 1987), energy consumption during daily activity (assume 20% of total caloric intake), specific dynamic action of food (thermic effect; 6%-10% of total caloric intake) and the energy consumed during physical training (based on the non-protein RER/RQ). Not surprisingly the HMRM group exhibited a dietary deficit of -579.37±164.9 Kilocalories of energy per day. This compared to a surplus of 210.33± 89.58 Kilocalories in TRM and 246.72±154.52 Kilocalories in CONM. This deficit was consistent and significantly greater than either TRM or CONM ($P<0.05$) within the HMR group over the course of the investigation (post measure = -483.97±218.47 Kilocalories). In contrast to the HMRM group HMRF appeared to consume more absolute calories than either TRF or CONF. HMRF consumed 28.5% more calories than TRF and 30% more than the CONF group. When body weight was factored into the caloric intake measure there was a clear difference in energy consumption. HMRF consumed an average of 42.85 Kcal/kg compared to 33.4 in TRF and 32.9 in the CONF group. It was interesting that HMRF consumed more calories per kilogram body weight than the HMRM group. The diet deficit/surplus values computed for the women subjects also demonstrated a value close to dietary equilibrium in all the groups. HMRF consumed a surplus of only +45.7 Kilocalories whereas TRF consumed +4.81 Kilocalories and CONF, +21.93 Kilocalories.

The dietary deficit of the HMRM group is interesting since the weight of this group remained stable over time despite a caloric deficit of 500 to 600 calories per day. A deficit of this magnitude would be expected to elicit a weight loss of at least one pound per week. According to accepted metabolic theory this would translate to a weight loss of 24 lbs over a six month period. Furthermore the greater diet deficit of the TRM group coincided with a slowing down of the weight loss effect of the exercise program.

This would suggest that the body must alter metabolically to compensate for an apparent diet deficit incurred due to a) inadequate nutritional intake b) increased energy expenditure due to running.

The commonly accepted practice of inducing weight loss by increasing energy expenditure and limiting dietary intake may not hold true for all cases since it has been demonstrated that reduction in food intake is associated with reductions in the BMR (Bray, 1969; Welle et al. 1984) and that the addition of exercise results in further reductions in metabolic rate (Warren, 1988; Phinney, 1985).

Dieting and manipulation of diet results in alterations in BMR. Bray (1969) demonstrated a 15% decrease in the BMR of a group of obese individuals after weight loss. Welle et al. (1984) demonstrated a 9.5% decrease in the BMR in response to a 472 kcal/day diet. Lammert and Hansen (1982) showed that semi-starvation resulted in a significant decrease in BMR compared to increases elicited by overeating. Although Stern (1980) suggested that exercise compensated for the decreased metabolic rate associated with semi-starvation others have demonstrated that caloric restriction and exercise have a three food greater suppression effect than food restriction alone (Phinney, 1985). Warren (1988) investigated the role of nutrition and energy balance in athletic amenorrhea in runners. The author reported that amenorrheic runners have significantly lower resting metabolic rates and thermic responses to food than normally cycling runners. Amenorrheic runners consumed a similar number of calories to normally cycling runners. She concluded that high mileage women runners did not increase caloric intake to compensate for activity but maintained energy balance through reductions in resting metabolic rate.

A decrease in basal metabolic rate would in part explain the apparent dietary deficit incurred by the HMRM and TRM and TRF groups since BMR was estimated for the purposes of this investigation. Besides a decrease in BMR the Warren finding of a decreased thermic effect of food suggests greater metabolic efficiency of digestion.

An increase in **food efficiency** (ratio of weight change to ingested calories , Brownell et al. 1987) occurs as a metabolic response to inadequate dietary intake. Body weight is maintained on fewer calories than would otherwise be expected.. A theory of increased food efficiency in runners is based on increased digestive efficiency for food and a reduction in metabolic rate. Leibell and Hirsch, (1984) reported that obese individuals required 28% less calories than normal weight individuals to maintain body weight. 25% less calories were required to maintain body weight after food restriction than prior to weight loss. Several investigations lend support for increased food efficiency in athletes. Drinkwater et al. (1984), Marcus et al. (1985) and Nelson et al. (1986) have reported relatively low caloric intake in athletes, despite high levels of energy expenditure, in sports where weight is important.

Alterations in metabolic rate and food efficiency may in part explain the apparent dietary deficit in the runners in the present investigation.

Alterations in dietary intake, metabolic rate, food efficiency and the motivational value of exercise:

An important aspect of the activity anorexia model is the increasing motivational value of exercise and reduction in the reinforcing value of food. The activity anorexia theory is based on a reduction of appetite induced by running which decreases the reinforcing effectiveness of food. As body weight declines (due to activity and decreased food intake) the reinforcing value of activity increases and further activity decreases food intake (Epling and Pierce, 1988). A theory of decreased metabolic rate and increased food efficiency with training is consistent with a model of activity anorexia.

If food efficiency increases in runners then body weight will be maintained on relatively lower caloric intake than prior to running and weight loss. If the runner uses running as a means of body weight control then further increases in running would be necessary to effect changes in body weight as the BMR decreases and food efficiency increases. Further desire for weight loss becomes a powerful motivator for further increases in running.

Evidence from the present investigation therefore supports a model of activity anorexia. Three important factors emerge:

1) Exercise does not result in increased energy intake.

2) The rate of change of daily running has a suppressing effect on food consumption.

3) Increasing activity is maintained against an increased relative caloric insufficiency.

Part 3: Endocrine alterations with exercise:

The hypotheses proposed were:

1) Six months of endurance training designed to increase the weekly training load of a group of healthy sedentary men and women to a mean of 40 to 48 km per week will result in a decrease in circulating testosterone in the men. Furthermore, high mileage runners will demonstrate significantly lower total testosterone levels than the sedentary male subjects.

2) Six months of endurance training will result in alterations in the pulsatile release of LH in the training group. Alterations will include decreased LH pulse frequency, decreased pulse amplitude and area under the LH curve. Furthermore, LH pulsatile characteristics will be significantly different between high mileage runners and sedentary but healthy men.

3) Alterations in testosterone will be related to alterations in LH pulsatile release in the training group.

4) Alterations in total testosterone and LH pulsatile characteristics will be related to caloric and macro-nutrient intake and changes in caloric intake.

Decreased total testosterone in HMR and TR at baseline and following training.

As previously reported by others (Wheeler et al. 1984; Strauss et al. 1985; Ayers et al., 1986) total testosterone levels were significantly lower in HMR versus the control subjects (pre-training group). Total testosterone levels were significantly reduced by six months of endurance training during which time the TRM increased their weekly training load to a mean of 39.5 km/week. Total testosterone levels fell by 22% from 885.4±59.1 to 684.1±58.3 ng/dl ($p<0.01$). As expected testosterone levels were 23% lower (885.4± 59.1 v 594.2± 58.1, $P<0.03$) in the HMR group versus the pre-training men. These findings are consistent with those of Wheeler et al. (1984) who reported total and free testosterone levels 30% lower in a group of 31 high mileage runners versus 18 sedentary controls and Ayers et al. (1986) who reported significantly reduced total testosterone levels in 14 out of a sample of 20 marathon runners. In the Ayers et al. investigation, T levels were 39% lower in the runners than the controls. Hackney et al. (1988) reported significantly reduced total and free testosterone levels in trained versus untrained men (499±46 v 725±67 ng/dl; 17.2±1.4 v 23.6±0.6 pg/ml, $P<0.001$). However, in the present investigation the Free Androgen Index (FAI; total T/SHBG ratio) was not significantly lower in the HMR v training group nor was it lowered by training. It should be noted that there was approximately a 50% difference in the FAI in HMR v TR and that large within group variation accounted for the lack of significance.

Others have attributed the fall in total and free testosterone to chronic training stress. Strauss et al. (1985) reported an 80% reduction in total testosterone from the beginning to the peak of the wrestling season (890±180 to 170±80 ng/dl).

This represents the first time that an endurance training program induced fall in total testosterone has been demonstrated, although several cross sectional investigations have suggested a decrease in total and free testosterone associated with endurance activities (Wheeler et al., 1984; Strauss et al., 1985).

Many different theories of mechanisms of decreased total and free testosterone associated with exercise have been proposed. To investigate the etiology of decreased testosterone, caloric intake and LH pulsatile release were examined in the groups over the six month training period.

The role of nutrition in decreased testosterone levels associated with exercise.

Although several authors have suggested a role of nutrient intake in alterations in the menstrual cycle in women (see previous discussion) there have been a lack of measurement of caloric intake in male runners with low testosterone levels. Several investigations have suggested a role of nutritional intake in reduced testosterone levels in runners. Ayers et al. (1986) suggested the presence of an anorectic sub-group in their investigation of marathon runners who demonstrated unusual emphasis on leanness and food intake. However, no measure of dietary intake was attempted. Strauss et al. (1985) suggested that a large reduction in caloric intake in a group of wrestlers was correlated with significant changes in male sex steroids. Although, undoubtedly, wrestlers are notorious for their abuse of nutritional common sense, Strauss and colleagues made no objective or statistical attempt to relate changes in testosterone to actual energy intake. Rather, this was inferred from large losses of body fat and body weight and significant correlations. Alterations in menstrual function are common in women runners and there is considerable evidence to suggest that alterations in dietary intake may in part be responsible. Menstrual abnormalities occur as a function of starvation in humans during times of social unrest (Stein et al. 1975). Malnutrition in pre-adolescents (Chakravarty et al. 1982; Kulin et al. 1984) and adults (Vigersky et al. 1977; Beumont et al. 1976) results in alterations in gonadotropin secretion. Others have suggested specific nutritional deficiencies are responsible for alterations in the menstrual cycle, including caloric deficiency (Drinkwater et al. 1984; Marcus et al. 1985; Nelson et al. 1986; Schweiger et al. 1988) excess dietary fibre (Lloyd et al 1987), protein deficiency (Schwartz et al. 1981), red meat deficiency (Brooks et al. 1984) and fat and zinc insufficiency (Deuster et al. 1963). Bates et al. (1982) examined 29 women with unexplained fertility and 18 with menstrual dysfunction. All these women were below ideal body weight. When 36 of the women followed a dietary regimen designed to increase body weight, 19 of the infertile women conceived spontaneously and 9 of 10 women with secondary amenorrhea resumed menstruation. The authors concluded that the practice of weight control may have been a cause of unexplained infertility and menstrual disorders in otherwise healthy women.

Dietary inadequacy and exercise will result in a net energy deficit. It is possible that in the face of this inadequacy that there is a shutdown of the reproductive axes in men and women to save energy. This is consistent with animals in the wild. Mating behaviour ceases during times of severe food shortage.

To relate changes in testosterone to various nutritional variables correlation coefficients were computed for absolute dietary intake and composition and relative dietary intake and composition (ie: relative to kg/body weight and kg/lean body mass). A stepwise regression analysis and factor analysis was also computed to examine potential relationships of nutritional variables and pre and post testosterone levels. (see results for tables of correlations).

Correlations for pre and post testosterone levels were computed for HMRM versus caloric intake, calories/kg body weight and other macronutrients. There was a significant relationship between caloric intake, calories/kg, calories/kg LBM, grams protein and fat/kg body weight with pre testosterone levels (all $p<0.01$). Post investigations correlation coefficients were significant for caloric intake/kg LBM, dietary protein/kg body weight and dietary fat (all $p<0.05$) and dietary protein/kg LBM ($p<0.01$). Although caloric intake and caloric intake/kg body weight were not significantly correlated with post testosterone levels, coefficients narrowly missed significance.

An extremely interesting trend appeared when the coefficients for pre and post .training were compared in the TR group. Correlation coefficients for pre testosterone levels and indices of dietary intake were inversely correlated prior to the investigation. Correlations were negative and significant. Caloric intake and caloric intake/kg body weight, and caloric intake/kg LBM were highly negatively correlated with total pre-testosterone ($r=-0.532$, $p<0.05$; $r=-0.69$, $r=-0.733$, $p<0.01$ respectively). Caloric deficit and dietary protein intake (absolute and relative) were also significantly inversely correlated. Following training this significant inverse effect was removed and correlations became positive yet not significant. This suggested a shift in the relationship of total testosterone and energy balance in men under training conditions.

Since there was a positive relationship of nutrient intake to testosterone in HMR and an inverse relationship in pre measures in the TR group, it was reasonable to expect a shift in the grouped correlation coefficients towards a positive relationship of calories and other nutrients (absolute and relative) to final testosterone levels. Indeed, examination of the coefficients revealed that following training there was a significant correlation of post testosterone to absolute and relative caloric intake, dietary deficit and absolute protein and dietary fat intake. These correlations suggested some interrelationship of testosterone levels and nutrient intake and may be reminiscent of a reduction in reproductive hormones in animals under starvation conditions. For example there was shift in the correlation coefficient of caloric intake, caloric intake/kg body weight and lean body mass and dietary deficit (absolute and per kg body weight from 0.22 to 0.5, $p<0.02$; 0.04 to 0.415, $p<0.05$; 0.06 to 0.42, $p<0.05$; 0.36 to 0.46, $p<0.05$ and 0.38 to 0.47, $p<0.02$ respectively.

A stepwise regression was computed to examine pre and post program predictors of total testosterone. The analysis of pre-investigation pooled data revealed no significant predictors of total testosterone. The post analysis revealed two predictors; caloric deficit/kg body weight and total caloric intake (all $P<0.05$).

Factor analysis also revealed some compelling evidence that testosterone was related to caloric intake and other nutrient factors. Pre analysis revealed 3 factors. Total calories and other measures loaded on factor 1, hereafter called the "diet factor." However, total testosterone and dietary deficit/kg/body weight were independent of this factor and were correlated with a third factor, the "hormone-energy" factor. Since the data was pooled and the HMR and TR groups both exhibited contrasting pre investigation relationships of nutrient intake and testosterone this was expected. The shift towards a relationship between the hormonal and diet factor was demonstrated by the significant correlation of total testosterone and diet deficit/kg body weight to the diet factor in the pooled factor analysis.

In summary the correlative data does suggest a significant relationship between dietary intake and total testosterone in those who exercise regularly and probably represents a physiological adjustment to increased energy expenditure without a compensatory increase in caloric intake. The exact significance of this remains unclear although conceivably such an adjustment may mimic periods of low food availability when reproductive activity ceases for energy conservation and survival.

The role of pulsatile LH secretion in the regulation of normal testicular production of testosterone.

LH is secreted in a pulsatile fashion from the anterior pituitary in men at approximately 90-140 minute intervals (Naftolin,Judd and Yen, 1973; Naftolin,Yen and Tsai, 1972).

The pulsatile secretion of LH from the anterior pituitary gonadotropes is necessary for the normal production of testosterone from the interstitium/Leydig cells. LH binds receptors at the Leydig cell site and promotes the conversion of 20-22 alpha-hydroxycholesterol to pregnenolone and eventually to testosterone. LH also plays a role in augmenting the membrane transport of the cholesterol precursor into the mitochondria for conversion to testosterone (Hafiez et al. 1971;1972; DiZerega and Sherins, 1981).

Research has demonstrated a functional link between the opiatidergic pathway and GnRH/LH pulsatile secretion. The administration of naloxone (opiate antagonist) increases basal LH levels and LH pulse frequency in women (see review). Subsequently, it has been suggested that increased opiatidergic tone might be responsible for an inhibition of GnRH and LH frequency with a concomitant reduction in peripheral production of testosterone. Current research has thus attempted to characterize the nature of LH pulsatile release in men and women who train over long distances.

Alterations in the pulsatile nature of LH have been demonstrated during acute exercise and at rest in chronically trained women endurance runners (Cumming et al. 1985,a & b). Such alterations in pulse frequency and amplitude may in part account for athletic amenorrhea. It has been suggested that similar alterations in men might account for a reduction in circulating testosterone levels commonly reported in chronically trained runners (Ayers et al.1985; Wheeler et al. 1984). However, evidence to date is contradictory with regard to this position. Rogol et al. (1984) reported no differences in LH pulse frequency or amplitude among runners and control subjects.

Hackney,Sinning and Bruot (1988) compared the hormonal profile of trained and untrained men and although total testosterone levels were significantly lower in the trained versus untrained group (499±46 v 725±67 ng/dl, $P<0.001$) there were no differences in LH pulsatile release or LH pulse amplitude. Basal LH levels were higher in the trained versus untrained group (15.3±1.9 v 11.7±1.2 mIU/ml, $P<0.05$). However, since the study was carried out with a four hour sampling period, a comparison of results is difficult. Maconnie et al. (1986) reported decreased LH pulse frequency in 6 healthy runners training an average of 120-200 km per week (2.2 v 3.6 pulses/8 hours, $p<0.05$). However, two hours of submaximal treadmill running failed to alter the LH secretory pattern in the running group. Runners also demonstrated an impaired LH response to increasing doses of exogenous GnRH. McColl et al. (1989, in press) reported that 60 minutes of exercise at 5% below the ventilatory threshold failed to alter LH pulsatile release in 6 highly trained men.

Futhermore, there is no evidence to support an opiate mediated reduction of LH pulse frequency in runners. Rogol et al. (1984) failed to show any difference in the NLX induced LH increment in trained versus untrained individuals. Elias et al. (1986) also failed to alter the LH response to exogenous LHRH stimulation with NLX in trained runners. A comparison of the investigations in which pulsatile parameters of LH secretion have been measured is reported below (see table 29). Comparison of LH pulsatile parameters from the various investigations is appropriate at this point.

Table 29. The Effects of Exercise and Opiate Antagonists on LH Pulsatile Release in Men.

Author	Subjects	Mean LH (mIU/ml)	LH Pulse Freq. (per 6/8/12 hr)	Amplitude (mIU/ml)	Periodicity (minutes)	Area under LHcurve (mIU/ml)
Naftolin et al. (1972)	Normal	NA	2-5/6hrs	5.6	60-100	NA
Veldhuis et al. (1983)	Normal	4.89	3.3/8hrs	3.7	135	2347
	+NTX	6.24	4.6/8hrs	3.0	96	2982
Veldhuis et al. (1984)	Normal	8.15	3.5/12hrs	NA	NA	4,600
	+NT[illegible]	9.69	6.0/12hrs	NA	NA	7,000
Rogol et al. (1984)	Runner	10.94	2.8/6hrs	4.36	144	5368
	Normal	10.53	3.18/6hrs	5.75	167	4098
	Runner+ NTX	13.58	4.9/6hrs	4.45	93.3	6511
	Normal+ NTX	14.26	4.64/6hrs	8.23	98.5	5892
MacConnie et al. (1986)	Runner	3.0	2.2/8hrs	0.90	NA	NA
	Normal	NA	3.6/8hrs	1.60	NA	NA
	Pre Run	3.0	1.8/8hrs	1.10	NA	NA
	Post 2hr run	NC	NC	NC	NC	NC
DeFeo et al. (1986)	Normals	6.6	3.3/6hrs	NA	NA	610
	+NLX	11.3	3.6/6hrs	NA	NA	1053
	Agonadal	36.6	4.2/6hrs	NA	NA	2344
	+NLX	38.6	3.6/6hrs	NA	NA	2205
McColl et al. (1988)	Runner Pre Ex	NA	4.0/6hrs	2.75	NA	1680
	Runner Post Ex	NA	3.6/6hrs	2.4	NA	1450
Wheeler et al (1988)	Normal	10.2	3.8/6hrs	4.41	102.2	NA
	@6mths	9.86	4.0/6hrs	4.15	78.75	NA
	HMR	11.42	3.33/6hrs	3.23	132.1	NA

LH Pulse Frequency.

Different sampling periods make it difficult to compare the investigations carried out to date on gonadotropin secretion in the high mileage runner.

Pro rating data would suggest that the data of McColl et al. (1988), Hackney et al. (1988) and the present investigation are consistent. The data from Rogol et al. (1984) and MacConnie et al. (1986) based on 8 hour sampling periods suggest a lower LH pulse frequency in trained men than that reported by the previous investigations. However, both groups of data are inconsistent with the LH pulse frequency reported by Veldhuis et al. (1984) in which only 3.5 pulses per 12 hour period were reported. LH pulses were elevated to 6.0/12 hours in the Veldhuis et al. (1984) investigation. This is approximately equivalent to the effect noted by DeFeo et al. (1986) and Rogol et al. (1984) with NLX and NTX administration respectively.

Mean LH Levels.

The present data is consistent with the data of Rogol et al. (1984) and Hackney et al. (1988). However, the data of MacConnie et al. (1986) would suggest very low baseline LH levels in high mileage runners training between 125-200 km/week.

Pulse Amplitude.

In comparison to other investigations the pulse amplitude reported by MacConnie would appear to be extremely low. McColl's data (1988) also appears to demonstrate LH pulse amplitude levels approximately 50% of those reported by Wheeler (present investigation), Rogol et al. (1984) and Hackney et al. (1988).

Periodicity.

The time interval between pulses across the various investigations is difficult to evaluate since the values reported in comparable investigations range from 78.75 to 144 minutes in runners training 80 Km/week.

Area under the LH curve.

Area under the LH curve is an estimate of the total LH secreted during a sampling period. The data from the MacConnie et al. (1986) investigation is not available for examination. However, it is interesting to observe that in subjects training a mean of 80km/week in the Rogol et al. (1984) and McColl et al. (1989) investigations that the values reported by the latter appear to be less than 50% of the former. Since the Rogol et al. (1984) investigation used samples following a 10-15 mile run than it is perhaps appropriate to compare the values to the McColl et al. (1988) investigation following 60 minutes of running at 5% below ventilatory threshold. The values from the McColl et al.

(1988) investigation are very much lower than those reported by Rogol et al. (1984). Values were not computed during the present investigation.

Summary:

There is a considerable variability in the research findings with regard to LH pulsatile release in runners under baseline and acute exercise conditions. the data from the MacConnie et al. (1986) investigation does appear to be somewhat discrepent from data reported by others.

It is therefore appropriate to examine the investigation conditions of previous studies on pulsatile gonadotropin release in men since there appears to be some inconsistency in research findings. A summary of investigation conditions is reported in table 30 below.

Table 30. A Comparison of Investigation Conditions of Gonadotropin Secretion in Trained Men and Control Subjects.

Variable	Author			
	Rogol et al. (1984)	MacConnie et al. (1986)	McColl et al. (1988)	Wheeler (1989)
Age of subjects	26-42	25.0	26	
Number subj. (runners)	n=25	n=5	n=6	n=6
Training state	80 km/wk	125-200/wk	80 km/wk	80 km/wk
Body fat	<10%	7.7%	N/A	
Season	Winter	Spring/Summer	Summer	Winter baseline Summer retest
Time of study	N/A	0600-1400	1100-1800	1100-1800
Duration of sampling	8/hrs	8/hrs	6/hrs	6/hrs
Alterations in LH Pulse freq (baseline & post exercise)	No diff. ruuners v controls after 10-15 mile run	Dec. LH pulse freq. runners v controls. Dec. response to GnRH in runners. No effect of 2 hour run @ 72% VO2 max.	No diff. LH pulse freq. runners after 60 min run @ 5% below Ventilatory threshold. No diff. v sedentary con.	No diff. @ baseline HMR v control No effect of 6 months endurance training
Pulse detect. method	Santen & Bardin (1973) Steiner(1982)	Reame et al. (1984)	Veldhuis et al. (1987)	Veldhuis et al (1987)
Testosterone (baseline)	Normal baseline No info. given	No difference runners v con. @ baseline	Low normal range v controls	Dec. total T HMR v Con Dec. total T @ 6 months training
Testosterone (Post ex.)	No Information	No effect of 2 hrs run @ 72% VO2 max.	Inc. T with 60 mins run @ 5% below Vent. Thresh	No acute ex phase

An examination of inter-investigation differences:

Possible explanations of discrepencies between investigations in which the integrity of the GnRH/LH pulse generator has been examined include:

a) Degree of training of subjects/training intensity..
b) Age of subjects.
c) Time/season of the investigation.
d) Duration of blood sampling.
e) LH pulse detection methodologies.
f) Effects of exercise; proximity to sampling period.
g) Variations in steroid levels and feedback mechanisms.
h) Body fat.
i) Nutritional state.

a) Degree of training of subjects:

It is highly evident from an analysis of the experimental conditions reported above that the runners in the MacConnie et al. (1986) investigation were training significantly more than the subjects in other investigations. It is also evident that this was the only investigation in which a significantly lower LH pulse frequency was reported in runners v control subjects. Baseline mean LH levels and LH pulse amplitude were also very different to those reported elsewhere. MacConnie et al. (1988) explained the discrepency between their data and that of Rogol et al. (1984) based on training volume. Rogol et al. (1984) on the other hand, observed that the absence of demonstrable abnormalities in LH pulse secretion in marathon runners should not be taken to completely exclude abnormalities in less strenuously training men just beginning training. The present investigation addressed this very issue. Six months of endurance training in previously sedentary men did not result in any changes in parameters of LH release.

Little information is given on the training intensity of the subjects in the various investigations under examination. The findings of unaltered LH pulse frequency with acute exercise reported by MacConnie et al. (1986) and McColl et al. (1988) under similar exercise intensity are comparable. Intensity variability between investigations would not appear to explain inter-investigation discrepencies.

b) Age of subjects.

The range of ages of subjects reported in the present investigation and Rogol et al. (1984) are consistent. Findings in the two investigations were similar with respect to LH pulsatile release. LH and T levels were not significantly correlated with age in the present investigation. The mean ages of subjects reported by MacConnie et al. (1986), Hackney et al. (1988) and McColl et al. (1989) were similar. However, whereas MacConnie reported significantly reduced LH pulse frequency in their runner subjects versus controls, the other investigations did not show any differences. The major difference between these investigations would appear to be training volume

c) Daily and seasonal timing of the investigations.

The MacConnie et al. (1986) investigation was begun early in the morning whereas those of other investigators were begun in the mid-morning. Since LH pulse frequency and testosterone are known to vary diurnally (Naftolin, Yen and Tsai, 1972) then this may possibly suggest a reason for variations in former investigation when compared to other studies.

It is impossible to pinpoint to exact season in which the investigations were carried out although it would appear that the studies of Wheeler (present), Rogol et al. (1984) and Hackney et al. (1988) were carried out in the winter. Results of the studies are comparable. The McColl et al. (1989) and MacConnie et al. (1986) investigations were carried out in the summer months yet there are different results. Again the degree of training of the MacConnie et al. (1986) subjects would appear to be the key variable.

d) Duration of sampling:

A comparison of investigations of alterations in pulsatile LH with exercise is difficult since the multiple blood sampling periods has ranged from 4 to 8 hours in trained subjects. To compare the investigations one can convert pulse frequencies to a standard frequency per sampling time. The sampling standard will be taken as 6 hours since this was the sampling duration chosen by our laboratory. These conversions are reported in table 31.

Table 31: Standardized pulse frequencies in exercise investigations:

Author	Subjects	Pulse frequency	Corrected (6 hours)
Rogol et al. (1984)	Runners	2.8/8 hours	2.1/6 hours
	Controls	3.2/8 hours	2.4/6 hours
MacConnie et al. (1986)	Runners	2.2/8 hours	1.7/6 hours
	Controls	3.6/8 hours	2.7/6 hours
	Runners (pre run)	1.8/8 hours	1.4/6 hours
	Post 2hr run	1.8/8 hours	1.4/6 hours
McColl et al. (1988)	Runners	4.0/6 hours	4.0/6 hours
	Post 60 min. run.	3.6/6 hours	3.6/6 hours
Hackney et al.(1988)	Runners	2.6/4 hours	3.9/6 hours
	Controls	2.7/4 hours	4.0/6 hours
Wheeler et al. (1989)	Runners	3.3/6 hours	3.3/6 hours
	Controls	3.8/6 hours	3.8/6 hours
	Post 6 mth training	4.0/6 hours	4.0/6 hours

A comparison of the pro rated data illustrates major differences between the data of Rogol et al. (1984) and MacConnie et al. (1986) versus the findings of McColl et al. (1988), Hackney et al. (1988) and the present investigation. The subjects utilized by McColl et al. (1988) and in the present investigation were comparable in terms of training distance although no information other than 5 years of training is given in the Hackney et al. (1988) investigation. The subjects used by Rogol et al. (1984) and MacConnie et al. (1986) were marathon runners although training distances were different between the two investigations. It should also be noted that the use of a standardized pulse frequency is a contrivance and does not necessarily reflect alterations in periodicity within individuals over time. It merely serves as a basis for comparison.

e) Total and free Testosterone levels and LH pulse frequency:

The presence of significantly reduced total and free testosterone (Wheeler et al. 1984) and total testosterone (Ayers et al. 1985) has been established in the high mileage runner. An examination of the investigations of gonadotropin release as an etiological factor in the reduction of sex steroids in endurance trained men casts doubt on this a mechanism.

In the present investigation significantly reduced total testosterone was observed in HMR v sedentary controls and after 6 months of endurance training in TRM. This data is consistent with that reported by Hackney et al. (1988) in which total and free testosterone were reduced in trained v untrained subjects (499±46 v 725±67 ng/dl, 17.2±1.4 v 23.6±0.6 pg.ml) without any alterations in LH pulse frequency compared to a group of control subjects.

However, Rogol et al. (1984) reported neither reduced total or free T or LH pulse frequency in men training 80km/wk. and MacConnie et al. (1986) reported significantly reduced LH pulse frequency in highly trained men without alterations in total testosterone. Although the LH response to GnRH was impaired in runners, an acute 2 hour bout of exercise at 72% of VO2 max. had no effect on pulse frequency. McColl et al. (1988) reported significantly reduced total T levels v control subjects but no difference in pulse frequency or alteration in pulse frequency with 60 minutes of running at 5% below ventilatory threshold. This data would not support a hypogonadotropism mediated decrease in testosterone production in men who run.

f) Body fat:

Alterations in the lean:fat ratio and decreased total body fat may be associated with the development of athletic amenorrhea (Cumming and Rebar, 1985). Anorectics with very low body fat become amenorrheic. Strauss et al. (1985) correlated low body fat with low testosterone levels in wrestlers and Ayers et al (1985) reported low body fat in an anorectic sub-group of ultra-distance runners associated with low total and free testosterone and oligospermia. Low body fat levels commonly associated with endurance running might conceivably result in hypogonadotropism and hypoandrogenism in men. Total testosterone and LH pulse frequency were not related in the present investigation. Body fat levels reported by Rogol et al. (1984), MacConnie et al. (1986) and Hackney et al. (1988) are comparable. Body fat levels were all less than 10% in the subjects within these investigations: yet LH pulse frequency is reduced in the MacConnie et al. (1986) study without decreases in total T; LH pulse frequency is not reduced in the Rogol et al. (1984)

investigation without changes in total testosterone and are unaltered in the Hackney et al. (1988) investigation in the presence of total and free T levels 70% of control values. Body fat would not appear to be a significant factor in between investigation variations.

g) Nutritional status of subjects.

No information is given with regard to the nutritional state of the athletes in MacConnie. et al. (1986), Rogol et al. (1984) or Hackney et al. (1988). In the present investigation a relationship of caloric intake and T levels was established although there was no apparent association with LH pulsatile release.

Summary statement:

Evidence from the present investigation would not support a theory of a centrally mediated suppression of testosterone production due to inhibition of the GnRH/LH axis. LH pulse frequency, amplitude and area under the curve was not significantly different between HMR and untrained healthy men. Although HMR had a marginally lower LH pulse frequency (3.3± 0.33 v 3.8± 0.37/pulses/6 hours) this difference was not significant. Furthermore, six months of endurance training failed to alter LH pulse frequency or any other aspect of the LH profile in the TR group, whereas a significant reduction in total testosterone did occur.

Several factors must however, be considered in evaluating this position:

a) periods of sampling across investigations have been inconsistent (4 to 8 hours) and therefore results may not be comparable. A four hour sampling period v an eight hour sampling period could conceivably produce different within subject pulse frequency values.

b) Alterations in LH levels and LH pulse frequency may be extremely subtle and not detectable within the sensitivity ranges of current assay procedures or within the sampling time frames used.

c) Wide interindividual and intraindividual variation and limited sampling procedures may create difficulties of generalization of results to whole populations of runners.

d) Wide interindividual variations in training intensity and volume have not been considered in the analysis of the integrity of the HPG axis in high mileage runners.

Hypoprolactinemia as a mechanism of testosterone suppression.

Prolactin is necessary for the normal production of testosterone in the Leydig cells of the testes (DiZerega and Sherins, 1981). Prolactin appears to sensitize the testicular LH receptor to LH (Bartke, 1976; Bartke and Dalterio, 1976; Hafiez, Lloyd and Bartke, 1972).

Wheeler et al. (1984) reported significantly reduced prolactin levels in high mileage runners whereas others (Hackney et al. 1988) have failed to report any changes or differences in PRL levels among trained and untrained subjects.

The present investigation revealed no significant differences in PRL levels between HMR and the TR group although there was a significant reduction in PRL with training in the TR group (11.6 ng/ml to 8.9 ng/ml, $p<0.05$).

Such reductions were within the physiological range and are unlikely to affect testosterone production at the testicular level.

Other mechanisms have been suggested to explain the reductions in circulating testosterone.

The fall in circulating testosterone levels must reflect either:

a) decreased production rates

b) decreased binding

c) increased metabolic clearance rate.

Production rates:

As previously discussed it would not appear that alterations in LH pulsatile release or prolactin levels are responsible for the decreased production of testosterone. Significant correlations of testosterone levels with nutrient factors imply a relationship but cannot be considered as causal (ie: a direct mechanism). Anecdotal evidence in the form of low lean body mass in endurance trained men suggests a decrease in protein synthesis or increased catabolism. Production rates of testosterone are decreased 10% in men after 60 minutes of sub-maximal endurance running (Cadoux-Hudson et al., 1985) although no chronic decrease in production rate of testosterone has been demonstrated in highly trained athletes.

The role of the ACTH-Cortisol axis in lower testosterone levels in trained men.

Insulin induced hypoglycemia induces a decrease in testicular production of testosterone presumably due to a direct inhibitory effect on the

Leydig cells (Cumming, Quigley and Yen, 1983). Chronic elevation of cortisol levels has been demonstrated in "overtrained" endurance athletes (Barron et al., 1985) and a shift in the testosterone/cortisol ratio associated with strenuous activity has been reported (Adlercreutz et al. 1986). Wheeler et al. (1984) reported normal values of cortisol in high mileage runners (15.9±1.8 ug/dl). Cortisol levels were not significantly correlated with total or free testosterone values. There was no difference in cortisol levels between high mileage runners and controls in the Wheeler et al. (1984) investigation. It is possible that elevated cortisol levels associated with endurance training might suppress normal testicular production of testosterone.

However, in the present investigation there was no difference in cortisol levels in HMR and TR and no effects of training on cortisol levels. All levels were within the normal range for afternoon samples for adult men.

Body Fat and Fat/Lean Ratio:

Alterations in body fat and the fat to lean ratio may in part be responsible for amenorrhea often reported in women athletes (Cumming and Rebar, 1985). Strauss et al. (1985) reported a significant relationship between reduced total and free testosterone and body weight, loss of body weight and percent body fat in a group of wrestlers. Ayers et al. (1986) reported pathologically reduced total and free testosterone levels in a sub-group of marathon runners with extremely low body fat.

Correlations of percent body fat, weight, weight loss and LBM with total testosterone (pre and post values) failed to reveal any relationship in the present investigation. However, it was interesting that an inverse correlation of body fat and total testosterone in the TR group became larger and significant with training ($p<0.05$). This would not support a decrease in testosterone associated with a decrease in body fat.

Since testosterone is metabolized by lean body tissue then it might reasonably be expected that testosterone might be associated with lean body mass. Since arguably runners defend much less lean body mass than non-runners then low testosterone levels might be expected in this group as a function of lower anabolic demand. However, there was no relationship between testosterone and lean body mass in any of the groups and HMR did not have significantly less lean body mass than the TR or CON groups.

Decreased testosterone binding capacity:

A decrease in binding of testosterone to SHBG is unlikely since in this and other investigations no alterations in SHBG capacity have been demonstrated (Wheeler et al. 1984). One investigation did report a decrease in SHBG in male marathoners but without a corresponding decrease in free T levels (Ayers et al. 1985)

Increased metabolic clearance of testosterone and reduced levels in athletes.

Since high mileage runners incur considerable structural damage associated with chronic training (see Hikida et al. 1983; Norregard-Hansen et al. 1982) then it might be expected that there is a greater peripheral demand and turnover of testosterone in runners versus sedentary individuals.

Skeletal muscle has specific androgen receptors (Snowchowski et al. 1981) and metabolizes testosterone in vitro (Stenstead and Eik-ness, 1981).

Testosterone prevents skeletal muscle degradation in rats undergoing strenuous endurance running. (Dahlman et al. 1981).

It is possible therefore, that continuous endurance training imposes an increased metabolic demand for testosterone at the peripheral muscular sites and that there is an increase in testosterone clearance analogous to increased thyroxine (T4) turnover in animals and man under strenuous exercise conditions (Irvine, 1967; 1968).

However, if utilization increases in the muscle then one would expect a compensatory increase in blood production rate of testosterone to compensate for increased removal (Wheeler et al. 1984). It has been demonstrated that free testosterone is correlated with metabolic clearance rate (Vermeulen et al. 1969). Reduced free T levels in other investigations would however, support an increased clearance theory (Wheeler et al. 1984; Hackney et al. 1988). However, although differences and changes in the FAI were not statistically significant they were marked and this mechanism must not be ruled out. Evidence from acute exercise based research has suggested that there is a decrease in metabolic clearance rate associated with an acute exercise induced rise in testosterone levels (Sutton et al., 1978; Cadoux-Hudson et al., 1985). To date it is unclear how MCR is affected by chronic endurance training.

In Summary:

The mechanism of reduced testosterone levels in high mileage runners is unclear. Alterations in LH pulse frequency do not appear to play a role in the reductions or are too subtle for current assay methodologies to discern. It does appear that caloric intake and other absolute and relative measures of dietary composition may be related to testosterone levels although caution must be exercised in interpreting correlation as cause. There is little doubt that dietary manipulation (Hill et al. 1980; Hamalainen et al., 193), starvation (Zubiran and Gomez-Mont, 1954) and anorexia nervosa (Brown, 1983) are related to severe hypogonadism. The alteration in hormonal levels and possibly testicular function has a survival value to both man and animals in times of low food availability. The exact mechanism is unclear by which energy balance regulates the HPG axis although likely reflects alterations in Pituitary-Thyroid function.

Conceivably a reduction in pituitary-thyroid function resulting in reduced metabolic rate might reduce the demand for testosterone by peripheral musculature. However, in the case of runners this would not seem likely due to excessive activity levels. It must be considered that the metabolic cost of exercising is relatively low for an endurance trained athlete and anecdotal evidence supports a considerable lethargy among runners during non-running periods of the day. Since it is known that the feeding centers of the hypothalamus are associated with the serotoninergic, dopaminergic and opiatidergic pathways (Morley and Levine, 1983) and that opiates and catecholamines have been implicated in the regulation of both appetite and the integrity of the HPG axis then it is in theory, possible, that the opiate pathway may influence testicular function based on the energy balance of the human organism. To date no investigation has demonstrated this relationship.

Energy balance and testicular function requires further examination in the long distance runner.

Chapter 5.

Conclusions and Recommendations.

Results of the investigation suggest that:

1) High mileage men and women runners do not consume more calories than sedentary individuals as compensation for increased energy expenditure even when gender and group differences in body weight and lean body mass are taken into account.

2) High mileage men runners appear to incur a caloric deficit due to training yet maintain a stable body weight. High mileage women runners also exhibit a deficit although not to the same extent as male runners.

3) As expected high mileage runners had a higher VO2 max. and, lower percent body fat than non runners (before a training program) and sedentary control subjects.

4) Six months of moderate intensity progressive endurance training resulted in significant increases in VO2 max. in men and women but a significant weight and body fat loss in training men only.

5) A six month program of moderate intensity (target heart rate zone) progressive endurance running training did not effect an increase in caloric intake to compensate for increased energy expenditure.

6) A six month program of moderate intensity progressive endurance running training did not result in qualitative dietary alterations in a group of healthy men and women.

7) A six month program of moderate intensity progressive endurance running resulted in an apparent dietary deficit in healthy men and women.

8) Individual dietary profiles suggested a gender difference in response to six months of moderate intensity progressive endurance running training. Men appeared to respond to running with a triphasic pattern of caloric intake. Women did not exhibit any significant trend in dietary intake. Men could be divided into responders (decreased caloric intake following onset of training) and non-responders (no decrease in caloric intake). Responders exhibited a significant initial decrease in caloric intake which did not recover to baseline and a significant quadratic and quartic trend in caloric intake. The significant trend affect was associated with a greater rate of increase in weekly running in the responder group versus non-responders.

9) High mileage male runners had significantly lower testosterone levels than sedentary men prior to six months of running training.

10) Six months of moderate intensity progressive endurance training resulted in a significant decrease in total testosterone in a group of previously untrained men. Pre experimental differences between high mileage male runners and sedentary men were removed after training.

11) Absolute and relative dietary intake factors were correlated with total testosterone in men who run.

In conclusion, the date supports a model of activity anorexia. Running training resulted in an apparent initial reduction in caloric intake. The effect was associated with the rate of change of weekly running. The effect appeared to be gender specific. Women did not exhibit any dietary trend as a result of training.

The data also supports recent reports of alterations in basal metabolic rate (BMR) and food efficiency (FE) with running training since high mileage runners maintained body weight on an apparent dietary deficit. Furthermore, healthy men and women training for six months failed to increase dietary intake to match the increased energy demands of exercise.

The potential alteration in physiological regulation of energy conservation may provide support for the motivational function of running expressed in the Epling and Pierce (1988) activity anorexia model. Since BMR may be reduced and FE increased with training, then the weight loss function of exercise may be lost as exercise progresses. Thus, as suggested in the Epling and Pierce (1988) model, the motivational value of running is increased and training volume and rate of change of activity increases.

Measurement of total testosterone in high mileage runners and a group of sedentary men prior to a six month training program confirmed earlier findings of significantly reduced levels in trained runners (eg.Wheeler et al.1984). The investigation demonstrates for the first time, a drop in testosterone levels associated with a controlled prospective investigation of running. The correlation of total testosterone with measures of energy intake suggests that energy balance is important in the explanation of alterations in the reproductive axis in men undergoing endurance training. The metabolic signal remains unexplained at this time. The following hypothetical model of the development of a condition of Activity Anorexia is proposed (figure 39). The model represents a modification of the Epling and Pierce (1988) model.

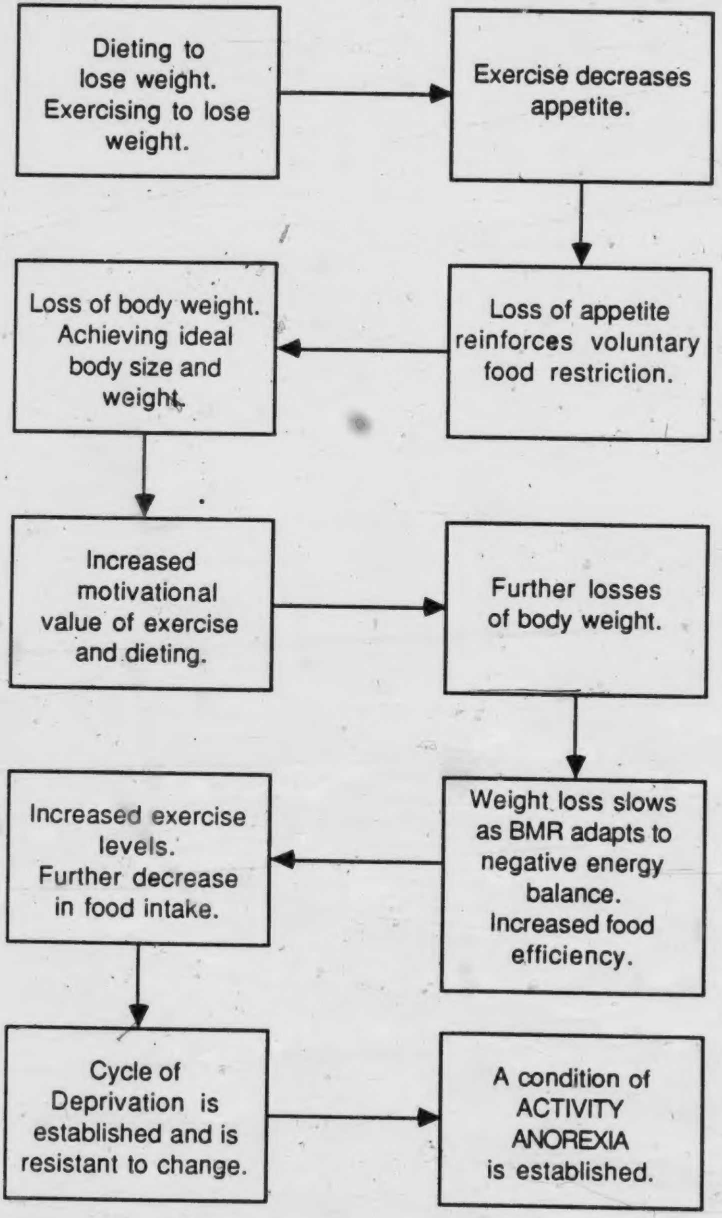
Recommendations for future research:

To examine the existence of a human activity anorexia a prospective investigation is required. Such an investigation would include following the progress of individuals who are engaging in exercise programs for recreational reasons and not for the purpose of a controlled laboratory investigation. In this manner the development and incidence of activity anorexia could be examined. Such an investigation would maximize the external validity of the activity anorexia construct.

To examine the role of energy balance in the regulation of the hypothalamic-pituitary-gonadal axis, the direction of future research must lie in:

a) Confirming the alteration in basal metabolism with exercise and training.
b) Identifying the metabolic signal responsible for altering HPG function.
c) Examining peripheral (muscular) uptake and utilization of testosterone.

Figure 39: A Hypothetical model of the deveopment of Activity Anorexia


Dieting to lose weight. Exercising to lose weight.
Exercise decreases appetite.
Loss of appetite reinforces voluntary food restriction.
Loss of body weight. Achieving ideal body size and weight.
Increased motivational value of exercise and dieting.
Further losses of body weight.
Weight loss slows as BMR adapts to negative energy balance. Increased food efficiency.
Increased exercise levels. Further decrease in food intake.
Cycle of Deprivation is established and is resistant to change.
A condition of ACTIVITY ANOREXIA is established.

(Reproduced with permission from "Activity Anorexia: A bio-behavioural perspective" Epling and Pierce (1988)

BIBLIOGRAPHY

Adlercreutz H, Harkonen M, Kuoppasalmi K, Naveri H, Huhtaniemi H, Tikkanen H, Remes K, Dessypris Karvonen J. Effect of training on plasma anabolic and catabolic steroid hormones and their response during physical exercise. Int. J. Sports Med. 7:27-28 (supp) 1986.

Ahrens RA, Bishop CL, Berdanier CD. Effect of age and dietary carbohydrate source on the response of rats to forced exercise. Journal of Nutrition. 102:241-248, 1972.

Appenzeller O, Standefer J, Appenzeller J. Neuorology of endurance training #5. Endorphins. (Abstract) Neurology. 30:418-419, 1980.

Appenzeller O. What makes us run? New Eng J Med. 305:578-579, 1981.

Ayers JWT, Komesu Y, Romain T, Ansbacher RA. Anthropometric, hormonal and psychologic correlates of semen quality in endurance trained male athletes. Fertil Steril. 43:917-921,1985.

Barron JL, Noakes TD, Levy W, Smith C, and Millar RP. Hypothalamic dysfunction in overtrained athletes. J Clin Endocrinol Metab. 60:803-806, 1985.

Bartke A. Pituitary-testis relationships. Role of Prolactin in the regulation of testicular function. In. Progress in Reproductive Biology of Sperm Action. Vol. 1:136. 1976. Kager. Basel.

Bartke A, and Dalterio S. Effects of prolactin on the sensitivity of the testes to LH. Biol. Reprod. 15:90-93, 1976.

Bates GW, Bates SR, and Whitworth, A. Reproductive failure in women who practice weight control. Fertil Steril., 37:373-378,1982.

Beaumont PJV, Berardwood C, Russel G. The occurrence of the syndrome anorexia nervosa in male subjects. Psychol. Med. 2:216-231, 1972.

Beaumont PJV, George GCW, Pimstone, BL. Body weight and the pituitary response to hypthalamic releasing hormones. J. Clin. Endocrinol. Metab. 43:1141-1145, 1976.

Bergeijk L van, Gooren LJG, van Kessel H, Sassen AM. Effects of naloxone on plasma levels of LH, FSH and in addition TSH and Prolactin in males before and after oestrogen or anti-estrogen treatment. Horm. Metab. Res. 18:611-615, 1986.

Blue A. Aerobic running as a treatment for moderate depression. Perceptual and Motor Skills. 48(1):228-231. 1979.

Blumenthal JA, O'Toole LC, Chang JL. Is running an analogue of anorexia nervosa? JAMA. 252:520-523,1984.

Blumenthal JA, Rose S, Chang JL. Anorexia nervosa and exercise. Med. Sci. Sports Exerc. 2:237-247, 1985.

Bonen A, Belcastro AN, Ling WY, Simpson AA. Profiles of selected hormones during menstrual cycles of teenage athletes. J. Appl. Physiol. Respirat. Environ. 50:69-85, 1981.

Boothby WM, Berkson J, Dunn HL. Studies of the energy of normal individuals: A standard for basal metabolism with a nomogram for clinical application. American Journal of Physiology. 116: 468, 1936.

Boothby WM. Handbook of Biological data: W.S. Spector (ed). 1956.

Bostick-Reed P. Nutrition: An applied science. West Pub. 1980.

Boyden TW, Pamenter RW, Stanforth PR, Rotkis TC, Wilmore JH. Impaired gonadotropin responses to gonadotropin releasing hormone stimulation in endurance trained women. Fertil Steril.41:359-363,1984.

Bray GA. Effect of caloric restriciton on energy expenditure in obese patients. Lancet. 2:397-398, 1969.

Brehm BA. Elevation of metabolic rate following exercise: Implications for weight loss. Sports Medicine. 6:72-78, 1988.

Brooks SM, Sanborn CF, Albrecht BH, Wagner WW. Diet in athletic amenorrhea. Lancet 4(1): 559-560, 1984.

Brown GM. Endocrine alterations in anorexia nervosa. In Garfinkel & Garner (eds.), "Anorexia nervosa: Recent developments in research." New York: Alan R. Liss Inc., pp. 231- 247, 1983.

Brown RS, Ramirez DE, Taub JM. The prescription of exercise for depression. Physician and Sports Medicine. 6(12):34-37, 1978.

Brownell KD, Steen SN, Wilmore JH. Weight regulation practices in athletes: an analysis of metabolic and health effects. Med. Sci. Sports. Exerc. 19:6 546-556, 1987.

Brozek J, Grande JT, Anderson T, Keys A. Densitometric analysis of body composition: revision of some quantitative assumptions. Ann NY Acad Sci. 110:113-140,1963.

Bruch H. Eating Disorders: Obesity, Anorexia nervosa and the person within. London, Poutledge&Kegan Paul. 1974.

Cadoux-Hudson TA, Few JD, Imms FJ. The effect of exercise on the production and clearance of testosterone in well trained young men. Eur J. Appl. Physiol. 54:321-325,1985.

Carr DB, Bullen BA, Skrinar GS, Arnold MA, Rosenblatt M, Beitins IZ, Martin JB, McArthur JW. Physical conditioning facilitates the exercise-induced secretion of beta-endorphin and beta-lipotropin in women. N Eng J Med . 305:560-563,1981.

Chakravarty I, Sreedhar R. Ghosh KK, et al.: Circulating gonadotropin profile in severe cases of protein calorie malnutrition. Fertil Steril. 37:650-654,1982.

Cjaja JA. Food rejection by female rhesus monkeys during the early menstrual cycle and during early pregnancy. Physiol and Behav. 14:579-587, 1975.

Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. Phys Rev. 57(4):779-815, 1977.

Clement DB, Taunton JE, Smart GW, McNicol KL. A survey of running injuries. Physician and Sports Medicine. 9(5):47-58, 1981.

Colt EWD, Wardlaw SL, Franz AG: The effect of running on plasma beta-endorphin. Life Sci. 28:1637-1640,1981.

Cornish ER, Mrosovsky N. Activity duirng food deprivation and statiation in 6 species of rodent. Animal Behaviour. 13:242-248, 1965.

Crews J, Aldinger EA. Effect of chronic exercise on myocardial function. Am Heart J. 74:536-547,1967.

Crews EL, Fuge KW, Oscai LB, Holloszy JO, Shank RE. Weight, food intake and body composition: Effects of exercise and protein deficiency. Am. J. Physiol. 216:359-363, 1969.

Crisp AH. Clinical and therapeutic aspects of anorexia nervosa. A study of 30 cases. J Psychosom Res. 9:67-78, 1965.

Crisp AH, Hsu, LKG, Harding B, Harthorne, J. Clinical features of of anorexia nervosa. A study of a consecutive series of 102 female patients. J. Psychosomatic Res. 24:179-191, 1980.

Cumming DC, Quigley ME, Yen SSC. Acute suppression of circulating testosterone levels by cortisol in man. J Clin Endocrinol Metab. 57: 671-673,1983.

Cumming DC, Rebar RW. Hormonal changes with acute exercise and with training in women. Sem Reprod Endcorinol. 3:55-64,1985.

Cumming DC, Vickovic MM, Wall SR, Fluker MR, Belcastro AN. The effect of acute exercise on pulsatile release of luteinizing hormone in women runners. Am J Obstet Gynecol. 153: 482-485,1985 (a).

Cumming DC, Vickovic MM, Wall SR, Fluker MR. Defects in pulsatile LH release in normally menstruating runners. J Clin Endocrinol Metab. 60: 810-812,1985 (b).

Cumming DC, Wheeler GD. Opiates in exercise physiology. Sem Rep Endocrinol. 5:171-179, 1987.

Cumming DC. The reproductive effects of exercise. Current Problems in Obstetrics and Gynecology. 10:231-285, 1987.

Dahlmann B, Widjaja A, Reinauer H: Antagonistic effect of endurance training and testosterone on alkaline proteolytic activity in rat skeletal muscles. Eur J Appl Physiol. 46:229-235, 1981.

Dale E and Goldberg E. Implications of nutrition in athletes' menstrual cycle irregularities. Can JAppl Spt Sci. 7(2):74-78, 1982.

Dally P. Anorexia Nervosa. NY, Grune and Stratton, 1969.

Dalvit SP. The effect of the menstrual cycle on patterns of food intake. Am J Clin Nutr. 34:1811-1815,1981.

Davis JM, Lamb DR, Lowy MT, Yim GKW, Malven PV. Opioid modulation of feeding behaviour following forced swimming exercise in male rats. Pharmacol Biochem Behav. 23:701-707,1985a.

Davis JM, Lamb DR, Yim GKW, Malven PV. Opioid modulation of feeding behaviour following repeated exposure to forced swimming exercise in male rats. Pharmacol Biochem Behav. 23:709-714,1985b.

DeFeo ML, Maggi M, Guardebasso V, Rodbard D, Delitala G, Fazzi V, Genazzani AD, facchinetti F, Forti G. Naloxone administration does not affect gonadotropin secretion in agonadal men either basally or during testosterone treatment. J. Clin. Endocrinol. Metab. 63(1):257-261, 1986.

Delitala G, Devilla, L, and Musso NR. On the role of dopamine receptors in the naloxone induced hormonal changes in man. J Clin Endocrinol Metab., 56: 181-184,1983.

Dempsey JA. Anthropometrical observations of obese and non-obese young men undergoing a program of vigorous physical exercise. Res. Q. 35:275-287, 1964.

Despres JP, Bouchard C,Tremblay A, Savard R, Marcotte M. Effect of aerobic training on fat distribution in male subjects. Med Sci Sports and Exercise.17(1):113-118, 1985.

Deuster PA, Kyle SB, Moser PB, Vigersky RA, Singh A, Schoomaker EB. Nutritional intakes and status of highly trained amenorrheic and eumenorrheic women runners. Fertil Steril. 46:636-643, 1986.

Di Zerega GS, Sherins RJ. Endocrine control of adult testicular function. The testes. Ed.Burger H, De Kreiser D, Raven Press. NY pp 127-140 N.Y., 1982.

Drinkwater BL, Nilson K, Chesnut CH, Bremner WJ, Shainoltz S, Southworth M. Bone mineral content of amenorrheic and eumenorrheic athletes. New Eng J Med. 311:277-281, 1984.

Edholm OG, Fletcher JG, Widdowson EM, McCance RA. Energy expenditure and food intake of individual men. Brit J Nutr. 9:286-300,1955.

Eik-Nes KB. Production and secretion of testicular steroids. Rec Prog Hormone Res. 27:517-535, 1971.

Ekblom B, Astrand PO, Saltin B, Stenberg J, Wallstrom B. Effects of training on the circulary repsonse to exercise. J Appl Physiol. 24:518-528, 1968.

Elias AN, Iyer K, Pandian MR, Weatherersbee P, Stone S, Tobis J. B-endorphin/B-Lipotropin release and gonadotropin secretion after acute exercise in normal males. J Appl Physiol Respirat Environ. 61(6):2045-2049, 1986.

Elliot DL, Goldberg L, Watts WJ, Orwoll E. Resistance exercise and plasma beta-endorphin/beta-lipotrophin immunoreactivity. Life Sci. 34:515-518,1984.

Epling WF, Pierce WD, Stefan L. Schedule induced self starvation: possible implications for anorexia nervosa. In: Bradshaw CM, Szabadi E, Lowe CF, (eds): Quantification of Steady State Operant Behaviour. Elsevier-N Holland Biomedical, Amsterdam, pp393-397, 1981.

Epling WF, Pierce WD, Stefan L. A theory of activity-based anorexia. Int J Eat Dis. 3:27-46, 1983.

Epling, W.F. and Pierce, W.D. Activity based anorexia in rats as a function of opportunity to run on an activity wheel. Nutr Behav. 2:37-49, 1984.

Epling WF, Pierce WD. Activity based anorexia: A biobehavioural perspective. Int. J. Eating Disorders. 17(4):475-485, 1988.

Farrel PA. Exercise and the endogenous opioids. N Eng J Med. 305:1591-1592,1981 (a).

Farrell PA, Gates WK and Maksud MG et al. Plasma B-Endorphin/B-Lipotropin immunocreactivity increases after treadmill exercise in man. (abstract) Med.Sci.Sports Exerc. 13(2):134, 1981 (b).

Farrell PA, Gates WK, Maksud MG, Morgan WP. Increases in plasma beta-endorphin/beta-lipotropin activity after treadmill running in humans. J Appl Physiol Respirat Environ. 52:1245-1259,1982.

Farrell PA. Exercise and endorphins - male responses. Med Sci Sports Exerc. 17:89-93, 1985

Feicht CB, Johnson TS, Martin BJ, Sparkes KE, Wagner WW Jr. Secondary amenorrhea in athletes. Lancet. 2:1145-1146, 1978.

Fichter MM and Pirke KM. Hypothalamic pituitary functioning starving healthy subjects. In KM Pirke and D Ploog (eds). "The psychobiology of anorexia nervosa." Berlin: Springer-Verlag, p.124, 1984.

Finger FW. The effect of food deprivation and subsequent satiation upon general activity in the rat. J Comp Physiological Psychol. 4:557-564, 1951.

Fishman SM, Carr DB. Naloxone blocks exercise stimulated water intake in the rat. Life Sci. 32:2523-2527,1983.

Frisch RE, McArthur JW. Menstrual cycles: fatness as a determinant of minimum weight for height necessary for their maintenance or onset. Science 185:949, 1974.

Frisch R, Gotz-Welbergen AV, McArthur JW, Albright T, Witschi J, Bullen B, Birnholtz J, Reed RB, Hermann H. Delayed menarche and amenorrhea of college athletes in relation to the onset of training. JAMA. 246:1559-1563,1981.

Garfinkel PE, Brown GH, Stancer HC, Moldofsky H. Hypothalamic pituitary function in anorexia nervosa. Arch Gen Psychiat., 32:734-744, 1975.

Garner, DM, Garfinkel PE. 'The Eating Attitudes Test: an index of symptoms of anorexia nervosa.' Psych Med. 9:273-279,1979.

Garner DM, Garfinkel PE, Olmstad MP. An overview of sociocultural factors in the development of anorexia nervosa.' In: Darby P, Garfinkel P, Garner DM, Coscina D eds. Anorexia nervosa: Recent developments in research. New York, NY: Alan Liss Inc. 65-82, 1983.

Garner DM and Garfinkel PE. Socioculturalfactors and anorexia nervosa. Lancet. 2:674, 1978.

Garner DM, Olmsted MP, Bohr Y, Garfinkel PE. The Eating Attitude Test: psychometric features and clinical correlates. Psychological Med., 12:871-878, 1982.

Glasser W. Positive addiction. Harper and Row, New York, 1976.

Grandison L, Guidotti A. Stimulation of food intake by muscimol and beta-endorphin. Neuropharmacology. 16:533-536,1977.

Greist JH, Klein MH, Eischens RR, Faris JT. Running out of depression. Phys Sports Med. 6(12):49-51,1978.

Greist A. Running as a treatment for depression. Comp.Psychiat., 20(1):41-54, 1979.

Hackney AC, Sinning WE, Bruot BC. Reproductive profiles of endurance trained and untrained males. Med Sci Sports Exerc. 2:60-65, 1988.

Hafiez A, Philpott JE, Bartke A. The role of prolactin in the regulation of testicular function: the effect of prolactin and luteinizing hormone on 3-B-hydroxysteroid dehydrogenase activity in the testes of rats and mice. J Endocrinol. 50:619-623, 1971.

Hafiez AA, Lloyd CW, Bartke A. The role of prolactin in the regulation of testes function: The effects of prolactin and luteinising hormone on the plasma levels of testosterone and androstenedione in hypophysectomised rats. J Endocrinol. 52:327-332, 1972.

Hall JF, Hanford PV. Activity as a function of a restricted feeding schedule. J Comparitive and Physiological Psychology. 47:362-363, 1954.

Hamalainen EK, Adlercreutz H, Puska P, Pietinen P. Decreases in serum total and free testosterone during a low fat high fiber diet. J Steroid Biochem. 18: 369-370, 1983

Henry SJP. The price of perfection. The Runner. 4(#6):35-39, 1982.

Hikida RS, Staron RS, Hagerman FC, Sherman WM, Costill DL. Muscle fiber necrosis associated with human marathoners. Journal of the Neurological Sciences. 59:185-203, 1983.

Hill P, Wynder E, Garbacrewski L, Games H, Walker ARP, Hellman P. Plasma hormones and lipids in men at different risk for coronary heart disease. Am J Clin Nutr. 33:1010-1018, 1980.

Holtzman SG. Effects of narcotic antagonists on fluid intake in the rat. Life Sci. 16:1465-1470,1975.

Holtzman SG. Suppression of appetite behaviour in the rat by naloxone: lack of effect of prior morphine dependance. Life Sci. 24:219-226,1979.

Howard RB. An inquiry into the morbid effect of deficiency of food chiefly with reference to their occurrence amongst the destitute poor. London England. Simpkin Marshall Co. 1939.

Irvine CHG. Effect of exercise on thyroxine degradation in athletes and non-athletes. J Clin Endocrinol Metab. 28:942-948, 1968.

Irvine CHG. Thyroxine secretion rate in the horse in various physiological states. J. Endocrinol. 39:313-320, 1967.

Kanarek RB, Collier GH. Patterns of eating as a function of the cost of the meal. Physiology and Behaviour. 23:141-145, 1979.

Kanarek RB, Collier, GH. Self starvation: A problem of overriding the satiety stimulus. Physiology and Behaviour. 30:307-316, 1983.

Kasch FW, Philips W, Carter JEL, Boyer JL. Cardiovascular changes in middle aged men during two months of training. J Appl Physiol. 34:57-59, 1973.

Katch FI, Michael ED, Jones EM: Effect of physical training on the body composition and diet of females. Res Quart. 40:99-103,1969.

Katch VL, Martin R, Martin J. Effects of exercise intensity on food consumption in the male rat. Am J Clin Nutr. 32: 1401-1407, 1979.

Katz JL. Long distance running, anorexia nervosa and bulimia: A report of two cases. Comprehensive Psychiatry. 27:1, 74-78, 1986.

Kaye WH, Pickar D, Naber D, Ebert MH. Cerebrospinal fluid opioid activity in anorexia nervosa. Am J Psychiatr. 139:643-645,1982.

Keys A, Brozek J, Henschell A, Mickelson O, Taylor HL. The Biology of Human Starvation. Minnesota, University of Minn. Press. 1950.

King A. Primary and secondary anorexia nervosa syndromes. Br J Psychiat. 109:470, 1963.

Kirkham TC, Blundell JE: Dual action of naloxone on feeding revealed by behavioural analysis: separate effects on initiation and termination of eating. Appetite. 5:47-54,1984.

Klibanski A, Beitins IZ., Badger T. et al. Reproductive function during fasting in men. J Clin Endocrinol Metab. 53:258-263,1981.

Knuth UA, Nieschlag E. Effect of different doses of naloxone on serum levels of prolactin and gonadotropins in young male volunteers. J. Enodcrinol. Invest. 8:55-57, 1985.

Kron L, Katz JL, Gorzynski G, Weiner H. Hypereactivity in anorexia nervosa: a fundamental clinical feature. Cmprehensive Psychiatry. 19:433-440, 1978.

Kulin HE, Bwibo N, Mutie D. Gonadotropin excretion during puberty in malnourished children. J. Pediatrics. 105: 325-328, 1984.

Kyung NH, Barkan A, Klibanski A, Badger T, McArthur JW, Axelrod L, Beitins IZ. Effect of carbohydrate supplementation on reproductive hormones during fasting in men.J Clin Endocrinol Metab. 50:827-835, 1985.

Lammert O, Hansen ES. Effects of excessive caloric intake and caloric restriction on body weight and energy expenditure at rest and light exercise. Acta Physiol scand. 114: 135-141, 1982.

Ledoux M, Brisson GR, Peronnet F. Nutritional status of adolescent female gymnasts. Med Sci Sports Exer. 14:145-154, 1982.

Leibell RL, Hirsch J. diminished energy requirements in reduced obese patients. Metabolism 33:164-170, 1982.

Leibowitz SF, Hor L, Endorphinergic and alpha-adrenergic systems in the paraventricular nucleus: effects on eating behaviour. Peptides 3:421-428,1982.
Leonardson, G.R. 'Relationship between Self concept and perceived physical fitness Percept Motor Skills. 44:62, 1977.

Levitsky D. Feeding conditions and internal relationships. Physiology & Behavior. 12:779-787, 1974.

Litt IF, Glader L. Anorexia nervosa, athletics and amenorrhea. Journal of Pediatrics. 9:1, 150-153, 1986.

Lion L.S. 'Psychological effects of jogging' Percept Motor Skills. 47:1215-1218, 1978.

Little JC. Neurotic iilness in fitness fanatics. Psych Abstracts. 667:49-56, 1981.

Lloyd T, Buchanan JR, Bitzer S. Interrelationships of diet, athletic activity, menstrual status and bone density among collegiate women. Am J Clin Nutr. 46:681-684, 1987.

Long BC. Aerobic conditioning and stress management innoculation: A comparison of stress management interventions. Cognitive Therapy and Research. 8(5):517-542, 1984.

Long BC. Stress management interventions: A 15 month follow up of aerobic conditioning and stress innoculation training. Cognitive Therapy and Research. 9(4): 471-478, 1985.

MacConnie SE, Barkan, A, Lampman, RM, Schork, MA, Beitins IZ. Decreased hypothalamic gonadotropin releasing hormone secretion in male marathon runners.N Eng J Med. 315:411-417, 1986.

MacDougall D, Wenger H, Green H. Physiological testing of the elite athlete. CASS, Mutual Press. 1982.

Maikel RP, Brauch MC, Zabik JE: The effects of various narcotic agonists and antagonists on deprivation induced fluid consumption.Neuropharmacology. 16:863-866,1977.

Malina RM, Spirduso WW, Tate C, Baylor AM. Age at menarche and selected menstrual characteristics in athletes at different competitive levels and in different sports. Med Sci Sports Exerc. 10: 218-222, 1978.

Malina RM. Menarche in athletes:a synthesis and hypothesis. Ann Human Biol. 10:1-24, 1983.

Marcus R, Cann C, Madvig P. Menstrual function and bone mass in elite women distance runners. Ann Int Med. 102:158-163, 1985.

Margules DL, Moisset B, Lewis MJ, Shibuya H, Pert CB: B-endorphin is associated with overeating in genetically obese mice (ob/ob) amd rats (fa/fa). . Science. 202:988-991,1978.

Mayer J, Marshall NB, Vitale JJ, Christensen JH, Mashanlkhi MB, Stone FJ: Exercise, food intake and body weight in normal rats and genetically obese adult mice. Ann J Physiol. 177:544-548,1954

Mayer J, Roy P, Mitra KP. Relation between caloric intake, body weight and physical work: studies in an industrial male population in West Bengal. Am J Clin Nutr. 4: 169-175, 1956.

McColl E, Wheeler G.D. Cumming DC..The effects of acute exercise on LH pulsatile release. Clinical Endocrinology (in press), 1989.

Mckay LD, Kenney NJ, Edens NK, Williams RH, Woods SC. Intracerebroventricular beta-endorpin increases food intake in rats. Life Sci. 29:1429-1434,1981.

Moore R, Mills IH, Forster A. Naloxone in the treatment of anorexia nervosa: effect on weight gain and lipolysis. J Roy Soc Med. 74:129-131,1981.

Morgan W.P.:Negative addiction in runners. Phys Sports Med. 7(2):56-70,1979.

Morley JE. Enodcrine effects of naloxone induced opiate receptor blockade. J Clin Endocrinol. 50(2):251-257, 1980.

Morley JE, Levine AS, Grace M, Kneip J: Dynorphin (1-13), dopamine and feeding in rats. Pharmacol Biochem Behav. 16:701-705,1982.

Morley JE, Levine AS: The central control of appetite. Lancet 398:401,1983.

Naftolin F, Yen SSC, Tsai CC. Rapid cycling of plasma gonadotropins in normal men as demonstrated by frequent sampling. Nature 236: 92-93, 1972.

Nelson ME, Fisher EC, Catsos PD, Meridith N, Turksoy N, Evans WJ. Diet and bone status in amenorrheic runners. Am J Clin Nutr. 43:910-916, 1986.

Norregard-Hansen K, Bjerre-Knudsen J, Brodthagen V, Jordal R, Pauley PE. Muscle leakage due to long distance training. Eur J Appl Physiol. 48:177-188, 1982.

Oscai LB, Holloszy JO. Effects of weight changes produced by exercise, food restricion or overeating on body composition. J Clin Invest. 48:2124-2128,1969.

Oscai LB. The role of exercise in weight control. Exer Sports Sci Rev. 1:103-123,1973.

Oscai LLB, Mole PA, Holloszy JO. Effects of exercise on cardiac weight and mitochondria in male and female rats. Am J Physiol. 220:1944-1948, 1971.

Pardridge WM. Transport of protein bound hormones into tissues in vivo. Endocr Rev. 2:103-123, 1981.

Pavlou KN, Steffee WP, Lerman RH, Burrows BA. Effects of dieting and exercise on lean body mass, oxygen uptake and strength. Med Sci Sports Exerc. 17(4):466-571, 1985.

Pfeiffer A, Braun S, Mann K, Meyer HD, Brantl V. Anterior pituitary hormone responses to a kappa opioid agonist in man. J Clin Enodcrinol Metab. 62(1): 181-185.

Phinney SD. The metabolic interaction between very low calorie diet and exercise. In Management of obesity by sever caloric restriction. G.L. Blackburn and G.A. Bray (eds). Littleton, MA. PSG Co. pp 99-105, 1985.

Pierce WD, Epling WF, Boer DP. Deprivation and satiation. The interrelationship between food and wheel running. J Exper Anal Behav. 46:199-210, 1986.

Pirke KM,Schweiger U,Lemmel W,Krieg JC and Berger M. The influence of dieting on the menstrual cycle of healthy young women. J Clin Endocrinol Metab. 60:1174-1179, 1985.

Plante RI and Houston ME. Exercise and protein catabolism in women. Ann Nutr Metab. 28:123-129, 1984.

Pollock ML. The quantification of endurance training programs. Exer Spts Sci Rev 1:155, 1973.

Premack D, Shaeffer RW. Some parameters affecting the distributional properties of operant level running. J Exper Anal Behav. 6:473-475, 1963.

Prior J.C.: Menstrual cycle changes with training: anovulation and short luteal phase. Can J Appl Spts Sci. 7:173-177,1982.

Prior JC, Ho Yuen B, Clement P, Thomas J. Reversible luteal phase changes associated with marathon training. Lancet. 1:269-270, 1982.

Prior JC, Cameron K, Ho Yuen B ,Thomas J. Menstrual cycle changes with marathon training. Anovulation and short luteal phase. Can J Appl Sports Sci. 7:173-177, 1982.

Quigley ME, Sheehan KL, Casper RF, Yen SSC.Evidence for increased dopaminergic and opioid activity in patients with hypothalamic hypogonadotropic amenorrhea. J Clin Endocrinol Metab. 50:949-952,1980.

Rogol AD, Veldhuis JD, Williams FA, Johnson ML: Pulsatile secretion of gonadotropins and prolactin in male marathon runners. J Androl. 5:21-27,1984.

Ropert J-F, Quigley ME, Yen SSC. Endogenous opiates modulate pulsatile luteinizing hormone release in humans J Clin Endocrinol Metab. 52:583-585,1981.

Rosen LW, McKeag DB, Hough DO, Curley V: Pathogenic weight control behaviour in female athletes. Phys Sportsmed. 14(1):79-86,1986.

Roskam H. Optimum patterns of exercise for healthy adults. Can Med AssocJournal 9:895-899, 1967.

Routtenberg A. Self starvation of rats living in activity wheels. Adapatation effects. J Comp.Physiological Psychology. 66:234-238, 1968.

Routtenberg A, Kuznesof AW. Self-starvation of rats living in activity wheels on a restricted feeding schedule. J Comp Physiol Psychol. 64:414-421,1967.

Russell Davis D. Studies in malnutrition. MRC special report series. London, HM stationary office. 1951.

Saltin B, Blomquist B, Mitchell JH, Johnson RL Jr, Wildenthal K, Chapman CB. Response to submaximal and maximal exercise after bed rest and training. Circulation. 38 (suppl. 7):1-78, 1968.

Saltin B, Rowell LB. Functional adaptation to physical activity and inactivity. Fed Proc. 39(5): 1506-1513, 1980.

Saltin B. Physiological effects of physical conditioning. Med Sci Sports Exerc. 1:50-56, 1969.

Sanger DJ, McCarthy PS. Differential effect of morphine on food and water intake in food deprived and freely feeding rats. Psychopharmacology. 72:103-106, 1980.

Schwartz B, Cumming DC, Riordan E, Selye M, Yen SSC, Rebar RW. Exercise associated amenorrhea: a distinct entity? Am J Obstet Gynecol. 141: 662-670, 1981.

Schweiger UF, Herman R, Lassle W. Caloric intake, stress and menstrual function in athletes. Fertil Steril. 49:447-450, 1988.

Short SH, Short WR. Four year study of university athlete's dietary intake. J. Amer Diet Assoc. 82:632-645, 1983.

Skinner JS, Holloszy JO, Cureton TK. Effects of a program of endurance exercises on physical work capacity and anthropometric measurements in 15 middle-aged men. Am J Cardiol. 14:747-752, 1964.

Smith NJ. Excessive weight loss and food aversion in athletes simulating anorexia nervosa. Pediatrics 66:139-142, 1982.

Snochowski M, Saartik T, Dahlberg E et al: Androgen and glucocorticoid receptors in human skeletal cytosol. J Steroid Biochem. 14: 765-771, 1981.

Sours JA. Running, anorexia nervosa and perfection. in Sachs and Sachs (eds): The Psychology of Running. Human Kinetic. Publ. 1981.

Spear NE, Hill WF. Methodological note: Excessive weight loss in rats living in activity wheels. Psychological Reports. 11:437-438, 1962.

Stanish W. Overuse injuries in athletes: a perspective. Med Sci Sports Exer. 16:1-7,1984.

Stenstad P, Eik-Nes KB: Androgen metabolism in rat skeletal muscle in vitro. Biochem Biophys Acta. 63: 169-176, 1981.

Stern JS, Schultz C, Mole P. Effects of caloric restriction and exercise on basal metabolism and thyroid hormone. Paper presented at the third International Congress of Obesity. October 8-11, Rome, Italy, 1980.

Stein Z, Susser M. Feritity,fecundity, famine: food rations in the Dutch famine 1944/45 have a causal relationship to fertility and probability to fecundity. Hum. Biol 47:131-154, 1975.

Stevenson JAF, Fox BM, Feleki V, Beaton JR: Bouts of exercise and food intake in the rat. J Appl Physiol Respirat Environ. 21:118-122,1966.

Strauss RH, Lanese RR, Malarkey WB: Weight loss in amateur wrestlers and its effect on testosterone levels. JAMA. 254:3337-3338, 1985.

Sutton JR, Coleman MJ, Casey JH. Testosterone production rate during exercise. In: F Landry and WAR Orban (Eds) 3rd International Symposium on Biochemistry of Exercise. Symposium Specialists, Miami. pp227-234, 1978.

Thomas TR, Adeniran SB, Etheridge GL. Effects of different running programs on VO2 max, percent fat and plasma lipids Can J Appl Sport Sci. 9(2):55-62, 1984.

Thompson DA, Wolf LA, Eikelboom R. Acute effects of exercise intensity on appetite in young men. Med Sci Sports Exerc. 20(3): 222-227, 1988.

Tokuyama K, Saito M, Okuda H. Effects of wheel running on food intake and weight gain of male and female rats. Phsyiology and Behaviour. 23:89-903, 1982.

Veldhuis D, Johnson ML. Cluster analysis: a simple, versatile, and robust algorithm for endocrine pulse detection. Am J Physiology. E486-493, 1986

Veldhuis JD, Rogol AD, Samojlik E, Ertel NH. Role of endogenous opiates in the expression of negative feedback actions of androgen and estrogen on pulsatile properties of luteinizing hormone secretion in man. J Clin Invest. 74:47-55., 1984.

Vermeulen A, Stoica T, Verdonck L. The apparent free testosterone concentration. An index of androgenicity. J Clin Endocrinol Metab. 33:759, 1971.

Vermeulen A, Verdonck L, Van Der Straeten M, Orie N. Capacity of testosterone binding globulin in human plasma and its influence on specific binding of testosterone and its clearance rate. J Clin Endocrinol Metab. 29:1470-1480, 1969.

Vigersky RA, Andersen AE, Thompson RH. Hypothalamic dysfunction in secondary amenorrhea associated with simple weight loss. New Eng J Med. 297:1141-1145, 1977.

Warren M. Delayed menarch in athletes: The role of low energy intake and eating disorders and their relation to bone density. International Symposium on Hormones and Sport. August 29-30, Dubrovnik, Jugoslavia. Abstract #19,1988.

Warren M., Brooks-Gunn, J. Scoliosis in ballet dancers. New Eng J Med. 314:1348-1353, 1986.

Watt EW, Wiley J, Fletcher G. Effect of dietary control and exercise training on daily food intake and serum lipids in postmyocardial infarction patients Am J Clin Nutr. 29:900-904,1976.

Weight LM, Noakes TD. Is running an analog of anorexia? A survey of the incidence of eating disorders in female distance runners. Med Sci Sports. Exerc. 19(3): 213-217, 1987.

Welle SL, Amatruda JM, Forbes GB, Lockwood DH. resting metabolic rate of obese women after rapid weight loss. J Clin Endocrinol Metab. 59:41-44, 1984.

Wheeler GD, Wall SR, Belcastro AN, Conger P, Cumming DC: Are anorexic tendencies prevalent in the habitual runner? Brit J Sportsmed. 20:77-81, 1986.

Wheeler GD, Wall SR, Belcastro AN, Cumming DC. Reduced serum testosterone and prolactin levels in male distance runners. JAMA. 252: 514-516, 1984.

Wood PD, Haskell WL, Blair SN. Increased exercise level and plasma lipoprotein concentration: A one year randomised controlled study in sedentary middle aged men. Metabolism. 32:31-39, 1983.

Yates A, Leehay K, Shisslak CM. Running: an analogue of anorexia. N Eng J Med. 308: 251-255, 1983.

Zubiran S. Gomez-Mont F. Endocrine disturbance in chronic human malnutrition. Vitamins and Hormones. 11:97-102, 1954.

APPENDIX 1.

RAW DATA: ALL GROUPS

	AGE	PREWT	MIDWT	POSTWT	PREFAT	MIDFAT	POSTFAT	PRELBM	MIDLBM	POSTLBM	PREVO2	MIDVO2	POSTVO2
1	50.000	58.100	60.000	58.100	16.100	16.100	15.400	51.400	51.000	52.500	54.700	56.300	62.800
2	49.000	69.100	71.800	71.000	12.000	10.800	8.600	60.950	63.500	65.180	54.500	57.500	63.000
3	42.000	60.900	60.000	60.500	12.900	12.800	13.300	52.200	53.500	53.600	61.900	66.800	66.900
4	50.000	71.800	71.000	70.900	24.500	21.700	21.500	56.100	56.200	55.600	56.200	56.700	59.500
5	53.000	63.600	62.500	60.900	10.100	10.000	9.500	57.300	56.860	56.600	52.000	52.400	59.900
6	48.000	70.000	68.200	70.900	22.600	26.600	26.600	56.500	51.600	52.700	51.400	52.400	57.400
7	56.000	63.300	61.000	60.000	22.400	16.500	16.900	48.900	49.400	51.300	44.700	51.700	59.100
8	29.000	60.000	60.000	60.000	10.200	7.600	6.000	53.500	55.400	57.100	78.300	78.300	79.200
9	54.000	61.000	62.000	61.000	17.700	15.300	17.500	52.000	52.360	51.100	44.300	46.000	52.800
10	41.000	76.400	75.900	76.400	7.900	10.800	6.000	72.400	70.200	71.100	62.800	57.500	64.100
11	44.000	55.900	54.500	54.000	29.400	21.500	24.600	40.200	42.100	41.100	46.300	38.400	42.700
12	27.000	50.200	51.200	51.600	18.600	18.850	19.100	40.860	41.870	41.000	50.900	50.700	51.600
13	23.000	52.300	52.300	51.400	17.200	17.200	22.800	42.500	41.800	40.500	60.400	64.900	61.600
14	23.000	57.700	58.200	56.400	17.000	18.300	18.800	47.200	47.100	46.450	56.000	54.800	49.700
15	23.000	53.600	54.500	53.600	11.100	12.500	13.400	47.500	48.000	46.220	59.200	56.500	66.600
16	43.000	48.200	47.700	46.400	21.900	15.700	15.200	37.450	40.300	38.600	56.100	57.300	51.300
17	23.000	46.400	47.300	46.800	13.200	13.600	13.900	39.800	40.890	40.000	58.500	58.500	61.800
18	40.000	83.400	83.800	82.800	19.200	10.700	12.000	68.000	73.400	72.800	42.600	49.000	47.800
19	41.000	73.300	73.500	71.000	22.300	22.900	20.700	56.900	55.900	52.700	35.300	38.300	46.100
20	49.000	65.500	66.100	63.500	23.500	22.900	21.900	50.140	50.400	50.300	41.700	45.200	49.600
21	24.000	70.600	70.600	70.600	12.900	11.100	12.800	60.600	61.800	59.500	48.100	55.000	55.100
22	33.000	93.400	91.000	87.000	27.200	13.900	12.400	81.600	79.040	77.700	37.800	44.100	46.500
23	21.000	79.400	81.100	77.200	18.100	13.400	14.700	65.040	68.400	65.400	40.100	50.000	55.100
24	30.000	96.200	90.600	88.000	26.900	18.300	15.700	79.500	74.500	75.200	37.400	50.300	50.900
25	28.000	76.800	76.400	71.300	21.200	14.900	13.900	60.5[illegible]	64.500	61.800	44.400	52.300	55.800
26	33.000	63.100	62.100	62.[illegible]00	15.000	14.900	16.600	53.[illegible]	52.800	50.000	44.100	46.800	51.100
27	25.000	71.000	70.000	71.000	8.200	12.100	10.200	62.400	61.600	63.000	49.500	59.300	61.400
28	21.000	71.500	72.300	71.600	15.900	16.500	16.600	59.950	61.200	58.400	44.300	57.800	58.000
29	42.000	81.000	80.500	78.000	22.000	19.800	19.600	63.200	64.900	61.700	40.000	48.200	45.000
30	28.000	81.900	80.000	76.400	14.400	15.900	14.800	68.800	67.800	66.400	39.600	51.900	50.700
31	45.000	69.600	67.200	67.700	18.000	12.900	16.400	56.700	58.000	56.600	49.700	50.900	52.200
32	32.000	69.900	68.400	64.900	27.500	25.200	24.600	50.680	51.300	50.680	31.200	,35.900	40.400
33	28.000	74.800	75.200	74.900	26.300	27.700	27.300	52.400	55.100	54.700	30.400	32.900	37.800
34	27.000	65.700	65.000	65.000	28.800	26.900	27.700	46.400	46.800	49.300	40.600	51.600	48.000
35	28.000	58.500	57.500	56.400	19.700	23.000	20.000	44.600	45.300	45.300	39.500	47.800	49.800
36	41.000	65.400	63.700	62.100	30.000	17.500	26.700	45.800	46.200	47.600	32.100	39.100	42.600
37	23.000	61.500	61.600	58.600	20.900	20.200	19.300	48.500	48.300	48.100	35.200	39.900	39.700
38	24.000	56.000	53.400	55.000	33.000	28.900	29.300	36.800	39.000	39.900	33.300	41.800	43.700
39	41.000	54.800	50.900	53.500	32.600	25.900	22.500	36.700	39.900	38.180	32.400	39.400	46.100
40	22.000	65.000	66.000	66.200	27.000	26.400	25.900	47.300	49.800	49.300	31.800	42.300	42.100
41	29.000	57.800	59.100	59.300	26.000	25.300	23.200	43.500	44.000	45.500	44.600	46.100	58.900
42	33.000	56.600	56.100	53.600	32.500	31.800	29.500	38.100	38.400	38.450	34.600	39.600	43.500
43	26.000	57.400	56.400	52.000	26.700	24.600	22.700	41.800	41.700	41.700	36.900	44.100	45.100
44	28.000	47.000	48.500	48.000	26.100	27.800	28.300	34.500	34.700	34.600	31.100	36.700	35.700
45	22.000	63.600	63.500	62.900	8.000	6.000	8.600	58.000	59.770	57.540	49.700	52.600	53.100
46	32.000	69.500	69.500	71.200	18.400	18.400	19.200	56.800	56.770	57.500	43.300	43.300	45.800
47	23.000	87.200	87.200	83.200	12.900	13.400	12.700	76.000	75.500	72.600	44.000	46.000	50.700
48	29.000	90.200	88.000	86.600	15.500	16.300	12.400	73.700	73.040	74.860	40.600	42.600	44.900
49	31.000	62.500	62.400	66.800	13.700	13.900	15.700	52.600	52.500	55.980	46.800	51.700	45.700
50	22.000	.	.	.	.	.	.	.	.	.	.	.	.
51	23.000	67.600	67.000	68.900	14.730	19.000	19.000	57.600	54.300	55.600	46.900	47.400	46.200
52	23.000	.	.	.	.	.	.	.	.	.	55.600	56.800	56.500
53	38.000	.	.	.	.	.	.	.	.	.	.	.	.
54	21.000	53.700	53.800	54.200	30.800	29.100	30.200	37.400	37.800	27.800	35.100	41.500	33.700
55	21.000	51.000	53.090	53.500	16.900	19.800	18.600	42.400	42.500	43.500	38.500	38.000	41.000
56	34.000	49.300	49.400	50.200	16.700	19.100	20.600	41.090	40.000	42.180	48.300	53.300	49.900
57	26.000	50.900	50.540	51.800	24.900	26.400	25.200	38.200	37.200	38.700	39.000	39.600	41.400
58	30.000	.	.	.	.	.	.	.	.	.	.	.	.
59	25.000	60.900	61.590	61.470	30.100	30.000	30.980	42.500	43.100	42.400	32.800	32.200	33.200
60	24.000	52.700	52.900	53.200	27.200	27.500	27.900	38.400	38.400	38.300	36.400	33.900	34.400
61	24.000	55.000	54.300	54.300	21.400	21.400	21.900	43.180	42.700	42.400	33.900	32.500	34.500
62	23.000	59.000	59.000	58.800	26.700	28.300	26.000	43.200	42.300	43.500	39.300	38.700	42.900
63	30.000	.	.	.	.	.	.	.	.	.	.	.	.
64	.	.	.	.	.	.	.	.	.	.	.	.	.
65	.	.	.	.	.	.	.	.	.	.	.	.	.

	PREDIS	MIDDIS	POSTDIS	PRECAL	MIDCAL	POSTCAL	PREGMSFAT	MIDGMSFAT	POSTGMSFAT	PREPROT	MIDPROT	POSTPROT
1	96.000	86.000	99.000	1962.000	1658.000	1580.000	80.000	43.000	54.000	78.000	70.000	62.000
2	24.000	37.000	24.000	2594.000	2801.000	1991.000	65.000	59.000	48.000	90.000	87.000	84.000
3	122.000	69.000	101.000	3627.000	2738.000	3005.000	134.000	113.000	129.000	128.000	119.000	110.000
4	56.000	64.000	86.000	2716.000	3110.000	2974.000	113.000	128.000	114.000	102.000	98.000	120.000
5	46.000	74.000	84.000	1954.000	2309.000	2430.000	80.000	87.000	98.000	75.000	92.000	86.000
6	94.000	74.000	40.000	2052.000	2372.000	2077.000	59.000	77.000	70.000	83.000	88.000	80.000
7	70.000	86.000	62.000	2143.000	1586.000	1805.000	108.000	71.000	90.000	76.000	84.000	85.000
8	75.000	66.000	60.000	2859.000	3330.000	3683.000	110.000	100.000	144.000	94.000	117.000	127.000
9	163.000	130.000	115.000	2495.000	2783.000	2681.000	89.000	80.000	94.000	91.000	71.000	72.000
10	96.000	66.000	115.000	2023.000	1780.000	1724.000	75.000	61.000	58.000	65.000	82.000	74.000
11	31.000	81.000	48.000	2274.000	2256.000	1656.000	62.000	82.000	49.000	89.000	84.000	63.000
12	30.000	30.000	32.000	2211.000	2340.000	2554.000	88.000	112.000	95.000	81.000	86.000	69.000
13	56.000	69.000	80.000	2922.000	2379.000	2916.000	96.000	66.000	70.000	118.000	88.000	105.000
14	42.000	37.000	51.000	2190.000	1927.000	1887.000	86.000	68.000	62.000	69.000	73.000	75.000
15	56.000	67.000	67.000	1971.000	1966.000	1471.000	57.000	64.000	36.000	73.000	67.000	51.000
16	26.000	47.000	45.000	2255.000	1989.000	2076.000	84.000	64.000	73.000	102.000	90.000	96.000
17	46.000	48.000	38.000	1792.000	1926.000	2330.000	54.000	55.000	74.000	80.000	78.000	77.000
18	0	23.000	29.000	2889.000	2384.000	2182.000	58.000	40.000	27.000	110.000	78.000	89.000
19	0	15.000	44.000	2375.000	1945.000	2498.000	105.000	98.000	114.000	91.000	71.000	84.000
20	0	25.000	29.000	2484.000	2233.000	2327.000	79.000	71.000	68.000	71.000	88.000	101.000
21	0	21.000	21.000	2385.000	3028.000	2596.000	147.000	125.000	99.000	122.000	106.000	98.000
22	0	20.000	55.000	2744.000	2266.000	2967.000	100.000	89.000	105.000	109.000	96.000	107.000
23	0	25.000	27.000	2363.000	3312.000	3062.000	115.000	141.000	113.000	128.000	147.000	122.000
24	0	29.000	40.000	3838.000	3649.000	3210.000	144.000	120.000	98.000	139.000	136.000	110.000
25	0	26.000	40.000	2826.000	3088.000	2796.000	125.000	148.000	95.000	93.000	90.000	120.000
26	0	38.000	43.000	2632.000	2430.000	2464.000	105.000	67.000	72.000	110.000	104.000	130.000
27	0	23.000	36.000	2475.000	2873.000	3384.000	80.000	81.000	105.000	105.000	105.000	113.000
28	0	28.000	47.000	2616.000	3194.000	3794.000	87.000	117.000	147.000	101.000	99.000	103.000
29	0	29.000	44.000	3066.000	2963.000	2550.000	91.200	113.000	83.000	145.400	89.000	93.000
30	0	21.000	50.000	2977.000	2986.000	2800.000	131.000	95.000	84.000	117.000	101.000	100.000
31	0	28.000	48.000	3038.000	2890.000	2922.000	105.940	100.000	99.000	118.750	131.000	132.000
32	0	21.000	29.000	2463.000	1596.000	1821.000	88.000	58.000	70.000	75.000	54.000	57.000
33	0	26.000	35.000	2890.000	2929.000	1489.000	148.000	115.000	67.000	81.000	88.000	64.000
34	0	28.000	50.000	1528.000	1791.000	2547.000	46.000	68.000	105.000	68.000	82.000	80.000
35	0	25.000	28.000	1835.000	2298.000	2212.000	57.000	78.000	73.000	80.000	75.000	88.000
36	0	15.000	32.000	1716.000	1668.000	1827.000	80.000	78.000	46.000	75.000	50.000	107.000
37	0	23.000	23.000	2967.000	2224.000	2301.000	93.000	91.000	84.000	67.000	80.000	78.000
38	0	23.000	32.000	1671.000	944.000	1468.000	58.000	26.000	42.000	70.000	42.000	54.000
39	0	21.000	47.000	1928.000	2323.000	2363.000	69.000	79.000	105.000	70.000	108.000	82.000
40	0	21.000	26.000	2289.000	2028.000	2295.000	71.000	60.000	86.000	71.000	85.000	88.000
41	0	31.000	49.000	2074.000	2022.000	2059.000	64.000	67.000	86.000	85.000	89.000	94.000
42	0	23.000	24.000	2367.000	2280.000	1938.000	99.000	99.000	83.000	81.000	82.000	70.000
43	0	23.000	42.000	1648.000	1986.000	1663.000	65.000	91.000	59.000	63.000	75.000	67.000
44	0	27.000	42.000	968.000	1035.000	1224.000	31.000	28.000	37.000	54.000	46.000	58.000
45	.	.	.	3315.000	2818.000	2599.000	102.000	102.000	95.000	109.000	95.000	84.000
46	.	.	.	2514.000	2414.000	2867.000	101.000	107.000	136.000	87.000	87.000	83.000
47	.	.	.	2498.000	3247.000	3401.000	129.000	122.000	113.000	82.000	90.000	95.000
48	.	.	.	2643.000	2570.000	2913.000	140.000	123.000	119.000	137.000	134.000	129.000
49	.	.	.	2174.000	2079.000	1848.000	87.000	77.000	71.000	82.000	79.000	89.000
50	.	.	.	.	.	.	.	.	.	.	.	.
51	.	.	.	2163.000	1973.000	2372.000	64.000	69.000	56.000	86.000	83.000	128.000
52	.	.	.	.	4032.000	2867.000	.	205.000	121.000	.	147.000	139.000
53	.	.	.	1363.000	1613.000	1812.000	68.000	66.000	62.000	51.000	39.900	34.000
54	.	.	.	2281.000	3323.000	2443.000	81.000	148.000	111.000	62.000	85.000	63.000
55	.	.	.	2195.000	2866.000	2238.000	89.000	108.000	96.000	82.000	94.000	76.000
56	.	.	.	2270.000	2400.000	3523.000	112.000	101.000	158.000	100.000	107.000	150.000
57	.	.	.	1227.000	1343.000	1706.000	51.000	59.000	51.000	47.000	57.000	49.000
58	.	.	.	1547.000	1989.000	2047.000	57.000	82.000	98.000	87.000	107.000	86.000
59	.	.	.	1583.000	1829.000	1704.000	64.000	85.000	63.000	76.000	71.000	57.000
60	.	.	.	2145.000	1566.000	1531.000	123.000	52.000	48.000	81.000	55.000	56.000
61	.	.	.	1397.000	1149.000	1245.000	50.000	42.000	48.000	52.000	41.000	44.000
62	.	.	.	2075.000	1778.000	1579.000	79.000	73.000	58.000	69.000	68.000	75.000
63	.	.	.	1502.000	1880.000	1627.000	46.000	44.000	50.000	57.000	82.000	61.000
64	.	.	.	.	.	.	.	.	.	.	.	.
65	.	.	.	.	.	.	.	.	.	.	.	.

	PRECHO	MIDCHO	POSTCHO	pre bmr	prebmr+activity	post bmr	caloric cost of activity	postBMR+ACT	predeficit
1	203.000	245.000	196.000	2005.600	2849.000	1967.400	871.500	2838.900	-887.000
2	372.000	385.000	273.000	2281.000	2513.400	2220.700	232.400	2453.100	80.600
3	432.000	257.000	276.000	2270.600	3468.700	2208.400	942.200	3150.600	218.300
4	247.000	320.000	317.000	3382.400	3990.400	2397.300	508.000	2905.300	-1174.400
5	232.000	302.000	320.000	1959.300	2373.600	2175.300	750.100	2925.400	-439.600
6	245.000	234.000	241.000	2181.700	2999.900	2184.200	348.200	2532.400	-947.900
7	232.000	172.000	187.000	2043.100	2569.600	1987.400	616.750	2604.150	-426.600
8	336.000	401.000	382.000	2250.200	2982.300	2333.600	590.200	2923.800	-123.500
9	327.000	437.000	411.000	2126.000	3314.100	2142.600	839.500	2982.100	-819.100
10	262.000	236.000	241.000	2303.300	3297.500	2273.400	1190.500	3463.900	-1274.500
11	337.000	304.000	227.000	1908.300	2164.300	1825.300	395.800	2221.100	109.700
12	295.000	262.000	353.000	1859.100	2072.000	1915.300	227.200	2142.500	139.000
13	369.000	369.000	369.000	1972.500	2455.100	1960.800	701.700	2662.500	466.900
14	281.870	255.510	258.288	1943.300	2309.600	1902.000	443.900	2345.900	-119.600
15	295.000	267.000	235.000	1910.400	2361.500	1860.400	540.500	2400.900	-390.500
16	291.000	274.000	269.000	1709.600	1894.100	1675.800	319.400	1995.200	360.900
17	250.000	280.000	330.000	1727.800	2038.200	1792.500	256.400	2048.900	-246.200
18	415.000	440.000	435.000	2710.580	2710.580	2603.130	324.000	2927.130	-21.580
19	226.000	179.000	210.000	2440.700	2440.700	2419.080	429.000	2848.080	-65.700
20	342.000	312.000	324.000	2337.800	2337.800	2254.400	278.000	2532.400	146.200
21	431.000	370.000	319.000	2605.300	2605.300	2626.200	224.000	2850.200	-220.300
22	245.000	239.000	341.000	2283.900	2283.900	2796.900	631.000	3427.900	460.100
2..	213.000	291.000	311.000	2633.300	2633.300	2691.200	296.600	2987.800	-270.300
24	449.000	465.000	456.000	2988.100	2988.100	2843.300	506.400	3349.700	849.900
25	325.000	350.000	363.000	2525.200	2525.200	2461.400	429.000	2890.400	300.800
26	270.000	303.000	299.000	2201.730	2201.730	2191.100	367.000	2558.100	430.270
27	283.000	347.000	420.000	2535.900	2535.900	2626.700	350.000	2976.700	-60.900
28	364.000	449.000	518.000	2615.500	2615.500	2628.000	490.000	3118.000	.500
29	409.256	421.000	332.000	2652.900	2652.900	2526.400	468.000	2994.400	415.100
30	247.000	335.000	322.000	2668.200	2668.200	2615.000	555.300	3170.300	308.800
31	331.127	375.000	380.000	2366.300	2366.300	2370.400	527.000	2897.400	671.700
32	293.000	214.000	206.000	2211.760	2211.760	2076.900	253.200	2330.100	251.240
33	310.000	408.000	167.000	2320.000	2320.000	2180.000	356.150	2536.150	570.000
34	213.000	226.000	317.000	2063.000	2063.000	2154.800	382.500	2537.300	-535.000
35	238.000	288.000	282.000	1930.700	1930.700	1946.600	210.000	2156.600	-95.700
36	131.000	145.000	102.000	2012.100	2012.100	1970.100	270.000	2240.100	-296.100
37	465.000	275.000	309.000	2207.600	2207.600	2108.100	155.200	2263.300	759.400
38	214.000	128.000	166.000	1880.400	1880.400	1855.100	242.000	2097.100	-209.400
39	226.000	232.000	239.000	1799.310	1799.310	1832.200	323.000	2155.200	128.690
40	306.000	289.000	307.000	2260.600	2260.600	2261.300	197.000	2458.300	28.400
41	255.000	230.000	239.000	2009.200	2009.200	2029.500	363.100	2392.600	64.800
42	283.000	163.000	225.000	2012.700	2012.700	1926.800	187.200	2114.000	354.300
43	185.000	223.000	174.000	1890.160	1890.160	1819.200	287.300	2106.500	-242.160
44	110.000	139.000	146.000	1704.000	1704.000	1727.600	232.000	1959.600	-716.000
45	514.000	390.000	350.000	2363.600	2363.600	2269.400	0	2269.400	951.400
46	229.000	235.000	267.000	2137.000	2137.000	2204.400	0	2204.400	377.000
47	266.000	422.000	482.000	2496.200	2496.200	2530.300	0	2530.300	1.800
48	203.000	231.000	316.000	2490.100	2490.100	2495.300	0	2495.300	152.900
49	287.000	257.000	216.000	2101.700	2101.700	2091.300	0	2091.300	72.300
50	.	.	.	.	.	.	0	.	.
51	296.000	282.000	341.000	2238.100	2238.100	2270.200	0	2270.200	-75.100
52	.	382.000	238.000	.	.	.	0	.	.
53	147.000	214.000	300.000	1684.600	1684.600	1729.500	0	1729.500	-321.600
54	317.000	434.000	308.000	1829.800	1829.800	1793.000	0	1793.000	451.200
55	271.000	320.000	272.000	1790.800	1790.800	1811.900	0	1811.900	404.200
56	205.000	232.000	363.000	1664.400	1664.400	1803.800	0	1803.800	605.600
57	138.000	151.000	276.000	1654.900	1654.900	1712.800	0	1712.800	-427.900
58	178.000	207.000	192.000	1701.300	1701.300	1761.500	0	1761.500	-154.300
59	188.000	208.000	241.000	1806.000	1806.000	1828.200	0	1828.200	-223.000
60	188.000	229.000	207.000	1781.300	1781.300	1730.000	0	1730.000	363.700
61	192.000	113.000	160.000	1716.600	1716.600	1438.600	0	1438.600	-319.600
62	257.000	214.000	191.000	1941.000	1941.000	1891.400	0	1891.400	134.000
63	226.000	308.000	173.000	1773.080	1773.080	1775.500	0	1775.500	-271.080
64	.	.	.	.	.	.	.	.	.
65	.	.	.	.	.	.	.	.	.

	predefratio	post deficit	postdefratio	testosterone2	SHBG2	cortisol2	prolactin2	FSH	POSTT2	postSHBG2
1	.689	-1258.900	.557	683.000	28.130	7.020	10.870	5.870	433.000	28.390
2	1.032	-462.100	.812	504.000	13.500	4.350	6.560	10.340	392.000	32.100
3	1.064	-145.600	.964	953.000	37.340	6.420	6.560	21.880	978.000	38.020
4	.898	88.700	1.024	730.000	48.000	3.880	24.090	12.760	796.000	47.400
5	.815	-495.400	.831	345.000	45.200	5.230	8.840	9.150	225.900	43.330
6	.684	-455.400	.820	393.000	15.200	6.800	7.580	4.990	731.000	23.500
7	.834	-799.150	.693	657.000	53.190	7.340	13.890	17.190	573.000	49.217
8	.959	769.200	1.263	700.000	63.400	6.720	8.520	15.900	795.000	32.130
9	.753	-321.100	.892	492.000	44.400	4.990	10.650	10.700	447.000	36.598
10	.613	-1739.900	.498	485.000	31.080	7.660	10.480	20.880	494.000	34.980
11	1.051	-565.100	.746	.	.	.	.	.	.	.
12	1.067	411.500	1.192	.	.	.	.	.	.	.
13	1.190	253.500	1.095	.	.	.	.	.	.	.
14	.948	-458.900	.804	.	.	.	.	.	.	.
15	.835	-929.900	.613	.	.	.	.	.	.	.
16	1.191	80.800	1.040	.	.	.	.	.	.	.
17	.879	281.100	1.187	.	.	.	.	.	.	.
18	.992	-745.130	.745	1036.000	32.220	8.030	15.020	7.140	545.000	30.100
19	.973	-350.080	.877	983.000	38.300	6.280	17.010	7.230	463.000	36.700
20	1.063	-205.400	.919	853.000	29.600	9.940	11.855	10.310	473.000	49.580
21	.915	-254.200	.911	946.000	28.020	10.740	10.595	5.230	958.000	23.700
22	1.201	-460.900	.866	1213.000	20.530	6.970	10.560	5.960	941.000	20.350
23	.897	74.200	1.025	833.000	11.860	8.750	13.970	6.160	847.000	11.900
24	1.284	-139.700	.958	569.000	14.850	8.006	15.020	3.530	439.000	11.030
25	1.119	-94.400	.967	1056.000	25.950	8.010	11.350	11.030	439.000	37.970
26	1.195	-94.100	.963	783.000	52.400	7.080	9.890	8.690	840.200	29.200
27	.976	407.300	1.137	1140.000	32.000	6.100	8.010	11.700	1015.000	29.580
28	1.000	676.000	1.217	867.000	41.900	7.770	8.670	9.880	798.000	38.460
29	1.150	-444.400	.852	538.000	33.830	3.250	11.320	5.880	571.900	43.700
30	1.116	-370.300	.883	1057.000	23.000	5.050	11.200	13.990	782.000	20.280
31	1.170	24.600	1.008	521.000	61.600	5.130	7.510	4.660	465.000	52.900
32	1.114	-509.100	.782	.	.	.	.	.	.	.
33	1.246	-1047.150	.587	.	.	.	.	.	.	.
34	.741	9.700	1.004	.	.	.	.	.	.	.
35	.950	55.400	1.026	.	.	.	.	.	.	.
36	.853	-413.100	.816	.	.	.	.	.	.	.
37	1.344	37.700	1.017	.	.	.	.	.	.	.
38	.889	-629.100	.700	.	.	.	.	.	.	.
39	1.072	207.800	1.096	.	.	.	.	.	.	.
40	1.013	-163.300	.934	.	.	.	.	.	.	.
41	1.032	-333.600	.861	.	.	.	.	.	.	.
42	1.176	-176.000	.917	.	.	.	.	.	.	.
43	.872	-443.500	.789	.	.	.	.	.	.	.
44	.580	-735.600	.625	.	.	.	.	.	.	.
45	1.403	329.600	1.145	.	.	.	.	.	.	.
46	1.176	662.600	1.301	.	.	.	.	.	.	.
47	1.001	870.700	1.344	.	.	.	.	.	.	.
48	1.061	417.700	1.167	.	.	.	.	.	.	.
49	1.034	-243.300	.884	.	.	.	.	.	.	.
50	.	.	.	.	.	.	.	.	.	.
51	.966	101.800	1.045	.	.	.	.	.	.	.
52	.	.	.	.	.	.	.	.	.	.
53	.809	82.500	1.048	.	.	.	.	.	.	.
54	1.247	650.000	1.363	.	.	.	.	.	.	.
55	1.226	426.100	1.235	.	.	.	.	.	.	.
56	1.364	1719.200	1.953	.	.	.	.	.	.	.
57	.741	-6.800	.996	.	.	.	.	.	.	.
58	.909	285.500	1.162	.	.	.	.	.	.	.
59	.877	-124.200	.932	.	.	.	.	.	.	.
60	1.204	-199.000	.885	.	.	.	.	.	.	.
61	.814	-193.600	.865	.	.	.	.	.	.	.
62	1.069	312.400	.835	.	.	.	.	.	.	.
63	.847	148.500	.916	.	.	.	.	.	.	.
64	.	.	.	.	.	.	.	.	.	.
65	.	.	.	.	.	.	.	.	.	.

	postcortisol2	post prolactin2	post FSH	Name	Group	Gender	Height	FTI Pre	FTI Post	PREEAt	POSTEAT
1	5.490	4.980	9.520	overton	1	male	160.500	84.306	52.958	3.00	3.00
2	4.280	5.350	10.140	campbell	1	male	166.600	129.630	42.402	2.00	1.00
3	7.280	8.000	32.250	whittinger	1	male	163.200	88.619	89.317	0	0
4	3.250	20.540	14.760	aarbe	1	male	176.400	52.807	58.310	5.00	3.00
5	5.560	5.770	12.190	germaine	1	male	171.500	26.503	18.102	5.00	3.00
6	6.630	7.570	6.980	muldowney	1	male	161.700	89.775	108.008	1.00	1.00
7	7.540	5.530	7.640	schotsky	1	male	161.700	42.897	40.425	3.00	2.00
8	5.530	7.340	23.840	rose	1	male	166.600	38.337	85.914	•	•
9	3.420	12.060	13.660	sedgrove	1	male	164.150	38.476	42.409	8.00	6.00
10	7.930	10.100	20.570	smith	1	male	166.800	54.184	49.036	15.00	4.00
11	•	•	•	mcfarland	1	female	161.700	•	•	2.00	1.00
12	•	•	•	viger	1	female	159.250	•	•	7.00	6.00
13	•	•	•	livingstone	1	female	161.700	•	•	17.00	13.00
14	•	•	•	leinhoff	1	female	156.800	•	•	5.00	5.00
15	•	•	•	kellal	1	female	161.700	•	•	5.00	6.00
16	•	•	•	hopchin	1	female	151.200	•	•	4.00	2.00
17	•	•	•	fedyma	1	female	154.300	•	•	7.00	7.00
18	3.810	10.290	3.630	gale	2	male	188.300	111.846	62.889	5.00	5.00
19	9.047	9.725	7.360	mcginnis	2	male	182.000	89.117	43.805	4.00	3.00
20	4.500	7.865	9.600	marsland	2	male	177.600	100.061	33.139	4.00	2.00
21	6.360	8.270	6.820	cooper	2	male	182.500	117.228	140.354	5.00	2.00
22	7.790	10.560	7.730	dodic	2	male	190.500	205.154	160.558	2.00	1.00
23	4.760	9.590	6.290	fisher	2	male	177.500	243.875	247.141	9.00	3.00
24	3.460	13.660	1.960	graham	2	male	187.500	133.043	138.196	3.00	1.00
25	4.950	6.331	8.150	henasyck	2	male	173.700	141.297	40.145	5.00	6.00
26	5.160	8.770	12.780	mcClure	2	male	162.500	51.885	99.910	7.00	10.00
27	5.760	10.010	11.460	kirby	2	male	181.000	123.698	119.225	1.00	4.00
28	6.650	10.410	8.900	wall	2	male	188.500	71.848	72.045	9.00	6.00
29	4.080	9.180	5.930	Barton	2	male	185.500	55.219	45.441	1.00	0
30	4.110	5.330	12.400	chalbern	2	male	180.200	159.571	133.889	3.00	2.00
31	4.420	6.300	2.020	schultz	2	male	174.700	29.367	30.521	25.00	22.00
32	•	•	•	veno	2	female	169.400	•	•	•	•
33	•	•	•	shaver	2	female	171.100	•	•	25.00	18.00
34	•	•	•	reimer	2	female	167.400	•	•	22.00	28.00
35	•	•	•	paradeen	2	female	160.800	•	•	33.00	16.00
36	•	•	•	mcginnis	2	female	165.900	•	•	30.00	12.00
37	•	•	•	murphy	2	female	173.100	•	•	13.00	7.00
38	•	•	•	day	2	female	160.020	•	•	5.00	4.00
39	•	•	•	blaeser	2	female	154.000	•	•	13.00	11.00
40	•	•	•	barry	2	female	182.300	•	•	6.00	14.00
41	•	•	•	askin	2	female	164.700	•	•	8.00	4.00
42	•	•	•	grant	2	female	168.000	•	•	3.00	3.00
43	•	•	•	hiron	2	female	155.500	•	•	6.00	5.00
44	•	•	•	mcclure	2	female	162.000	•	•	28.00	24.00
45	•	•	•	woudstra	3	male	169.050	•	•	4.00	4.00
46	•	•	•	welycochy	3	male	164.150	•	•	0	1.00
47	•	•	•	wreimer	3	male	170.275	•	•	3.00	1.00
48	•	•	•	ingham	3	male	171.500	•	•	•	•
49	•	•	•	albright	3	male	171.500	•	•	1.00	5.00
50	•	•	•	mcdonald	3	male	169.100	•	•	•	•
51	•	•	•	hornford	3	male	170.250	•	•	0	0
52	•	•	•	kennedy	3	male	173.950	•	•	•	•
53	•	•	•	samachy...	3	female	156.800	•	•	8.00	5.00
54	•	•	•	sokoluk	3	female	159.250	•	•	0	2.00
55	•	•	•	searle	3	female	161.700	•	•	1.00	2.00
56	•	•	•	samuels	3	female	149.450	•	•	6.00	5.00
57	•	•	•	richardson	3	female	160.000	•	•	1.00	1.00
58	•	•	•	olson	3	female	156.800	•	•	2.00	2.00
59	•	•	•	mish	3	female	159.400	•	•	3.00	7.00
60	•	•	•	harbin	3	female	157.800	•	•	8.00	4.00
61	•	•	•	gaunt	3	female	155.300	•	•	1.00	1.00
62	•	•	•	hagen	3	female	164.150	•	•	1.00	4.00
63	•	•	•	cooper	3	female	166.000	•	•	4.00	23.00
64	•	•	•		•		•	•	•	•	•
65	•	•	•		•		•	•	•	•	•

	PREEDI	POSTEDI	TRAINING INTEN PRE	TRAIN INT MID	TRAIN INT POST	SEX
1	•	•	•	•	•	1
2	8.00	5.00	•	•	•	1
3	•	•	•	•	•	1
4	•	•	•	•	•	1
5	5.00	3.00	•	•	•	1
6	10.00	5.00	•	•	•	1
7	•	•	•	•	•	1
8	•	•	•	•	•	1
9	8.00	6.00	•	•	•	1
10	9.00	2.00	•	•	•	1
11	15.00	13.00	•	•	•	2
12	14.00	8.00	•	•	•	2
13	2.00	2.00	•	•	•	2
14	•	•	•	•	•	2
15	10.00	9.00	•	•	•	2
16	3.00	3.00	•	•	•	2
17	7.00	7.00	•	•	•	2
18	8.00	6.00	15.500	16.500	16.500	1
19	8.00	6.00	11.250	12.250	13.250	1
20	2.00	5.00	14.000	14.750	15.250	1
21	17.00	6.00	14.000	16.000	16.000	1
22	3.00	3.00	12.000	13.000	14.250	1
23	32.00	21.00	13.500	13.750	14.250	1
24	10.00	8.00	12.250	14.750	15.250	1
25	29.00	27.00	13.250	15.000	15.750	1
26	1.00	8.00	14.000	14.250	15.500	1
27	6.00	8.00	15.000	17.250	18.000	1
28	34.00	28.00	12.500	16.000	16.500	1
29	9.00	12.00	14.750	14.750	14.750	1
30	9.00	6.00	13.500	14.000	15.500	1
31	3.00	8.00	13.500	13.500	13.500	1
32	•	•	10.500	11.000	11.000	2
33	38.00	31.00	11.000	11.250	11.250	2
34	38.00	53.00	14.250	14.750	15.000	2
35	39.00	27.00	12.500	13.250	13.750	2
36	72.00	14.00	10.500	12.500	12.600	2
37	19.00	32.00	11.250	11.000	12.000	2
38	24.00	22.00	10.750	11.500	11.500	2
39	16.00	6.00	12.000	13.000	13.500	2
40	30.00	51.00	12.000	13.000	13.000	2
41	19.00	26.00	12.750	13.500	14.000	2
42	4.00	2.00	10.500	11.000	12.000	2
43	37.00	13.00	10.000	11.000	12.750	2
44	28.00	44.00	11.000	11.500	12.050	2
45	4.00	10.00	•	•	•	1
46	1.00	0	•	•	•	1
47	5.00	5.00	•	•	•	1
48	•	•	•	•	•	1
49	9.00	9.00	•	•	•	1
50	•	•	•	•	•	1
51	2.00	6.00	•	•	•	1
52	•	•	•	•	•	1
53	9.00	9.00	•	•	•	1
54	8.00	7.00	•	•	•	2
55	6.00	6.00	•	•	•	2
56	9.00	15.00	•	•	•	2
57	4.00	3.00	•	•	•	2
58	3.00	2.00	•	•	•	2
59	25.00	33.00	•	•	•	2
60	7.00	12.00	•	•	•	2
61	2.00	2.00	•	•	•	2
62	3.00	3.00	•	•	•	2
63	38.00	82.00	•	•	•	2
64	•	•	•	•	•	•
65	•	•	•	•	•	•

APPENDIX 2A.

MEANS, STANDARD DEVIATIONS AND STANDARD ERROR FOR ALL VARIABLES BY GROUP: HIGH MILEAGE RUNNERS.

	VARIABLE	SEX	PRE MEAN	PRE STAND DEV	PRE SEM	MID MEAN	MID STAND DEV	MID SEM	POST MEAN	POST STAND DEV	POST SEM
1	AGE	M	47.20	7.98	2.93	•	•	•	•	•	•
2	AGE	F	29.43	9.73	3.68	•	•	•	•	•	•
3	HEIGHT	M	165.89	4.92	1.56	•	•	•	•	•	•
4	HEIGHT	F	158.09	4.16	1.57	•	•	•	•	•	•
5	WEIGHT	M	65.42	5.69	1.80	65.24	5.93	1.88	65.07	6.44	2.04
6	WEIGHT	F	52.04	4.07	1.54	52.24	3.91	1.48	51.46	3.71	1.40
7	BODY FAT (%)	M	15.64	5.95	1.88	14.80	5.81	1.84	14.23	6.70	2.12
8	BODY FAT (%)	F	18.34	6.01	2.27	16.79	3.14	1.88	18.26	4.36	1.65
9	LBM	M	56.13	6.70	2.12	56.00	6.38	2.02	56.68	6.54	2.07
10	LBM	F	42.22	3.81	1.44	43.15	3.08	1.16	41.98	3.09	1.17
11	VO2 (mls/kg/min)	M	56.08	9.91	3.13	57.56	9.06	2.87	62.47	7.06	2.23
12	VO2 (mls/kg/min)	F	55.34	5.05	1.91	54.44	8.26	3.12	55.04	8.46	3.20
13	CALORIC INTAKE	M	2440.50	535.89	169.46	2446.70	612.42	193.67	2394.00	682.40	215.79
14	CALORIC INTAKE	F	2230.73	351.74	132.94	2111.86	203.83	77.04	2127.14	509.65	192.63
15	FAT INTAKE (GMS)	M	91.30	24.00	7.59	81.90	25.96	8.21	89.70	32.84	10.39
16	FAT INTAKE (GMS)	F	75.57	17.47	6.60	73.00	18.98	7.18	65.57	19.03	7.19
17	PROTEIN INTAKE (GMS)	M	90.00	15.97	5.05	91.80	16.73	5.29	90.00	21.63	6.84
18	PROTEIN INTAKE (GMS)	F	87.43	17.30	6.54	80.57	8.64	3.27	79.43	18.81	7.11
19	CARBOHYDRATE INTAKE (GMS)	M	289.00	74.17	23.45	299.90	86.99	27.51	285.40	75.42	23.85
20	CARBOHYDRATE INTAKE (GMS)	F	302.69	38.81	14.68	287.26	39.26	14.84	291.61	58.07	21.95
21	TRAINING DISTANCE (KMS)	M	84.20	39.69	12.55	77.40	23.72	7.50	77.60	31.49	9.96
22	TRAINING DISTANCE (KMS)	F	41.00	12.39	4.69	54.40	18.59	7.02	51.57	16.68	6.31
23	TRAINING INTENSITY (KM/HR)	M	•	•	•	•	•	•	•	•	•
24	TRAINING INTENSITY (KM/HR)	F	•	•	•	•	•	•	•	•	•
25	BASAL METABOLIC RATE (PER 24 HRS)	M	2280.32	406.13	128.43	•	•	•	2189.00	135.37	42.81
26	BASAL METABOLIC RATE (PER 24 HRS)	F	1861.57	103.72	39.20	•	•	•	1847.44	94.49	35.71
27	BMR + ACTIVITY (PER 24 HRS)	M	3019.87	469.11	148.35	•	•	•	2877.97	300.00	94.87
28	BMR + ACTIVITY (PER 24 HRS)	F	2184.97	199.58	74.44	•	•	•	2259.57	230.67	87.18
29	CALORIC COST OF ACTIVITY (PER 24 HRS)	M	•	•	•	•	•	•	688.94	288.23	91.15
30	CALORIC COST OF ACTIVITY (PER 24 HRS)	F	•	•	•	•	•	•	412.13	167.66	63.37
31	CALORIC SURPLUS/DEFICIT	M	-579.37	521.51	164.92	•	•	•	-483.97	680.86	218.47
32	CALORIC SURPLUS/DEFICIT	F	45.74	314.21	118.76	•	•	•	-132.43	514.95	194.63
33	CALORIC SURPLUS/DEFICIT RATIO	M	.81	.16	.05	•	•	•	83	22	07
34	CALORIC SURPLUS/DEFICIT RATIO	F	1.02	14	.42	•	•	•	95	22	08
35	TESTOSTERONE (NG/DL)	M	594.20	183.57	58.05	•		•	586.49	231.21	73.12
36	CORTISOL (NG/ML)	M	6.04	1.32	.10	•	•	•	5.67	1.64	52
37	PROLACTIN (NG/ML)	M	10.98	5.02	1.58	•	•	•	8.72	4.72	1.49
38	FSH (mIU/ml)	M	12.97	5.86	1.85	•	•	•	15.16	8.09	2.56
39	LH (mIU/ml)	M	11.42	2.28	.93	•	•	•	•	•	•
40	SHBG (NMOL/L)	M	37.94	16.10	5.09	•	•	•	36.57	8.20	2.59
41	FREE T INDEX	M	64.55	32.37	10.20	•	•	•	58.69	27.38	8.66
42	LH PULSE FREQUENCY (PER 6 HRS)	M	3.33	.82	.33	•	•	•	•	•	•
43	AREA UNDER LH CURVE	M	•	•	•	•	•	•	•	•	•
44	EATING ATTITUDES	M	6.20	4.56	1.52	•	•	•	3.00	1.81	60
45	EATING ATTITUDES	F	7.00	4.86	1.82	•	•	•	5.71	3.90	1.48
46	EATING DISORDER INVENTORY	M	8.00	1.87	84	•	•	•	4.20	1.64	74
47	EATING DISORDER INVENTORY	F	8.50	5.47	•	•	•	•	7.00	4.05	1.65

APPENDIX 2C

MEAN DATA BY GROUP: CONTROLS

	VARIABLE	SEX	PRE MEAN	PRE STAND.DEV.	PRE SEM	MID MEAN	MID STAND.DEV.	MID SEM	POST MEAN	POST STAND.DEV.	POST SEM
1	AGE	M	25.63	4.27	1.51	•	•	•	•	•	•
2	AGE	F	26.73	5.08	1.53	•	•	•	•	•	•
3	HEIGHT	M	169.97	2.83	1.00	•	•	•	•	•	•
4	HEIGHT	F	158.79	4.47	1.35	•	•	•	•	•	•
5	WEIGHT	M	73.43	12.14	4.95	72.93	11.64	4.75	73.27	9.48	3.87
6	WEIGHT	F	54.06	4.07	1.44	54.33	4.09	1.45	54.68	3.69	1.31
7	BODY FAT (%)	M	13.87	3.45	1.41	14.50	4.74	1.94	14.60	4.15	1.70
8	BODY FAT (%)	F	24.34	5.50	1.94	25.20	4.40	1.56	25.17	4.50	1.59
9	LBM	M	62.45	9.82	4.01	61.98	9.86	4.03	62.35	8.89	3.63
10	LBM	F	40.80	2.42	.86	40.50	2.44	.86	39.85	5.27	1.86
11	VO2 (mls/kg/min)	M	46.73	4.97	1.88	48.63	5.25	1.98	48.99	4.50	1.70
12	VO2 (mls/kg/min)	F	37.91	4.84	1.71	38.71	6.83	2.41	38.88	5.94	2.10
13	CALORIC INTAKE	M	2551.17	421.67	172.14	2733.29	717.92	271.35	2695.29	489.23	184.91
14	CALORIC INTAKE	F	1780.45	409.90	123.56	1957.82	626.21	188.81	1950.46	621.56	187.41
15	FAT INTAKE (GMS)	M	103.83	27.65	11.29	115.00	44.73	16.91	101.57	29.02	10.97
16	FAT INTAKE (GMS)	F	74.55	25.41	7.70	78.18	31.70	9.56	76.64	35.20	10.61
17	PROTEIN INTAKE (GMS)	M	97.17	21.98	8.97	102.14	26.95	10.19	106.71	24.23	9.16
18	PROTEIN INTAKE (GMS)	F	69.46	17.13	5.17	73.36	23.89	7.20	68.27	30.97	9.34
19	CARBOHYDRATE INTAKE (GMS)	M	299.17	110.98	45.31	311.29	82.76	31.28	315.71	89.28	33.74
20	CARBOHYDRATE INTAKE (GMS)	F	209.73	53.97	16.27	239.09	81.38	26.35	243.91	64.95	19.58
21	TRAINING DISTANCE (KMS)	M	•	•	•	•	•	•	•	•	•
22	TRAINING DISTANCE (KMS)	F	•	•	•	•	•	•	•	•	•
23	TRAINING INTENSITY (KM/HR)	M	•	•	•	•	•	•	•	•	•
24	TRAINING INTENSITY (KM/HR)	F	•	•	•	•	•	•	•	•	•
25	BASAL METABOLIC RATE (PER 24 HR.	M	2304.45	172.21	70.31	•	•	•	2310.15	170.34	69.54
26	BASAL METABOLIC RATE (PER 24 HR.	F	1758.53	85.15	25.67	•	•	•	1752.38	116.01	34.98
27	BMR + ACTIVITY (PER 24 HRS)	M	2304.45	172.21	70.31	•	•	•	2310.15	170.34	69.54
28	BMR + ACTIVITY (PER 24 HRS)	F	1758.53	85.15	25.67	•	•	•	1752.38	116.01	34.98
29	CALORIC COST OF ACTIVITY (PER 2.	M	•	•	•	•	•	•	•	•	•
30	CALORIC COST OF ACTIVITY (PER 2.	F	•	•	•	•	•	•	•	•	•
31	CALORIC SURPLUS/DEFICIT	M	246.72	378.50	154.52	•	•	•	356.52	396.79	161.99
32	CALORIC SURPLUS/DEFICIT	F	21.93	376.50	113.39	•	•	•	198.07	585.79	176.55
33	CALORIC SURPLUS/DEFICIT RATIO	M	1.11	.16	.07	•	•	•	1.15	.17	.07
34	CALORIC SURPLUS/DEFICIT RATIO	F	1.01	.22	.07	•	•	•	1.11	.33	.10
35	TESTOSTERONE (NG/DL)	M	•	•	•	•	•	•	•	•	•
36	CORTISOL (NG/ML)	M	•	•	•	•	•	•	•	•	•
37	PROLACTIN (NG/ML)	M	•	•	•	•	•	•	•	•	•
38	FSH (mIU/ml)	M	•	•	•	•	•	•	•	•	•
39	LH (mIU/ml)	M	•	•	•	•	•	•	•	•	•
40	SHBG (NMOL/L)	M	•	•	•	•	•	•	•	•	•
41	FREE T INDEX	M	•	•	•	•	•	•	•	•	•
42	LH PULSE FREQUENCY (PER 6 HRS)	M	•	•	•	•	•	•	•	•	•
43	AREA UNDER LH CURVE	M	•	•	•	•	•	•	•	•	•
44	EATING ATTITUDES	M	1.60	1.82	.81	•	•	•	2.20	2.17	1.41
45	EATING ATTITUDES	F	3.18	2.93	.88	•	•	•	5.09	6.24	1.88
46	EATING DISORDER INVENTORY	M	4.20	3.11	1.39	•	•	•	6.00	3.94	2.12
47	EATING DISORDER INVENTORY	F	10.36	11.12	3.35	•	•	•	15.82	23.71	•

APPENDIX 2B.

MEAN DATA BY GROUP: TRAINING GROUP

	VARIABLE	SEX	PRE MEAN	PRE STAND.DEV.	PRE SEM	MID MEAN	MID STAND.DEV.	MID SEM	POST MEAN	POST STAND.DEV.	POST SEM
1	AGE	M	32.85	9.14	2.44	•	•	•	•	•	•
2	AGE	F	29.39	6.04	1.67	•	•	•	•	•	•
3	HEIGHT	M	180.86	7.47	1.99	•	•	•	•	•	•
4	HEIGHT	F	165.71	7.60	2.11	•	•	•	•	•	•
5	WEIGHT	M	76.91	9.71	2.56	76.01	8.93	2.39	74.02	7.82	2.09
6	WEIGHT	F	60.80	7.28	2.02	60.14	7.49	2.08	59.19	7.29	2.02
7	BODY FAT (%)	M	18.91	5.38	1.44	15.73	3.97	1.06	15.58	3.39	.91
8	BODY FAT (%)	F	27.47	4.08	1.13	25.48	3.69	1.02	25.15	3.39	.94
9	LBM	M	63.35	8.91	2.38	63.87	8.27	2.21	62.65	8.70	2.33
10	LBM	F	43.62	5.69	1.58	44.65	5.79	1.61	44.82	6.13	1.77
11	VO2 (mls/kg/min)	M	42.47	4.50	1.20	49.94	5.49	1.46	51.81	4.79	1.28
12	VO2 (mls/kg/min)	F	34.90	4.35	1.21	41.32	5.12	1.42	44.12	5.93	1.64
13	CALORIC INTAKE	M	2750.71	395.24	105.63	2802.93	480.00	128.29	2825.14	439.72	117.52
14	CALORIC INTAKE	F	2028.00	558.81	154.99	1932.54	539.19	149.54	1939.00	401.49	111.35
15	FAT INTAKE (GMS)	M	105.22	25.58	6.84	100.43	29.75	7.95	93.50	27.40	7.32
16	FAT INTAKE (GMS)	F	73.00	29.20	[illegible]	72.23	25.60	7.10	69.46	21.27	5.90
17	PROTEIN INTAKE (GMS)	M	111.51	19.51	[illegible]	103.07	21.68	5.79	107.29	14.74	3.93
18	PROTEIN INTAKE (GMS)	F	72.31	8.50	2.34	73.54	19.68	5.46	77.46	15.99	4.44
19	CARBOHYDRATE INTAKE (GMS)	M	325.03	80.04	21.39	348.29	81.37	21.75	359.29	77.45	20.70
20	CARBOHYDRATE INTAKE (GMS)	F	250.69	93.58	25.96	227.69	77.03	21.37	221.39	68.88	19.10
21	TRAINING DISTANCE (KMS)	M	0	0	0	25.07	5.44	1.45	39.50	9.58	2.64
22	TRAINING DISTANCE (KMS)	F	0	0	0	23.62	3.97	1.10	35.31	9.62	2.67
23	TRAINING INTENSITY (KM/HR)	M	13.50	1.19	.32	14.70	1.39	.37	15.30	1.27	.34
24	TRAINING INTENSITY (KM/HR)	F	11.46	1.19	.33	12.17	1.24	.34	12.65	1.18	.33
25	BASAL METABOLIC RATE (PER 24 HR	M	2540.39	203.60	54.41	•	•	•	2546.66	189.25	50.78
26	BASAL METABOLIC RATE (PER 24 HR	F	2023.19	185.90	51.56	•	•	•	1991.40	160.03	44.38
27	BMR + ACTIVITY (PER 24 HRS)	M	2540.39	203.60	54.41	•	•	•	2966.32	251.22	67.14
28	BMR + ACTIVITY (PER 24 HRS)	F	2023.19	185.90	51.56	•	•	•	2257.45	182.26	50.55
29	CALORIC COST OF ACTIVITY (PER 2	M	0	0	0	•	•	•	416.66	117.62	31.43
30	CALORIC COST OF ACTIVITY (PER 2	F	0	0	0	•	•	•	266.05	72.53	20.12
31	CALCRIC SURPLUS/DEFICIT	M	210.33	335.19	89.58	•	•	•	-141.18	363.62	97.18
32	CALCRIC SURPLUS/DEFICIT	F	4.81	419.29	116.29	•	•	•	-318.45	360.35	116.29
33	CALCRIC SURPLUS/DEFICIT RATIO	M	1.07	.12	.03	•	•	•	.95	.12	.03
34	CALORIC SURPLUS/DEFICIT RATIO	F	.99	.21	.06	•	•	•	.86	.21	.06
35	TESTOSTERONE (NG/DL)	M	885.36	220.97	59.06	•	•	•	684.08	217.95	58.25
36	CORTISOL (NG/ML)	M	7.22	1.99	.53	•	•	•	5.35	1.61	.43
37	PROLACTIN (NG/ML)	M	11.57	2.79	.75	•	•	•	8.99	2.12	.57
38	FSH (mIU/ml)	M	7.96	3.04	.81	•	•	•	7.50	3.45	.92
39	LH (mIU/ml)	M	10.19	.68	.30	•	•	•	9.86	1.57	•
40	SHBG (NMOL/L)	M	31.86	13.60	3.63	•	•	•	31.10	12.93	3.46
41	FREE T INDEX	M	116.64	59.12	15.80	•	•	•	97.66	62.65	16.74
42	LH PULSE FREQUENCY (PER 6 HRS)	M	3.80	.89	.37	•	•	•	4.00	.71	•
43	AREA UNDER LH CURVE	M	•	•	•	•	•	•	•	•	•
44	EATING ATTITUDES	M	5.93	6.04	1.61	•	•	•	4.79	5.61	1.50
45	EATING ATTITUDES	F	16.00	10.95	3.16	•	•	•	12.17	8.20	2.37
46	EATING DISORDER INVENTORY	M	12.21	11.34	3.03	•	•	•	10.86	8.24	2.20
47	EATING DISORDER INVENTORY	F	30.33	16.99	4.90	•	•	•	26.75	16.62	4.79

APPENDIX 3
RAW DATA: CALORIC INTAKE
MEN AND WOMEN

APPENDIX 3.

RAW DATA: NUTRIENT INTAKE: RELATIVE TO BODY WEIGHT: ALL GROUPS

	AGE	Name	Group	Gender	Height	SEX	precal/kg	midcal/kg	postcal/kg	vo21abs	vo22abs	vo23abs
1	50.000	everton	1	male	180.500	1	33.199	27.833	26.734	3.233	3.378	3.711
2	48.000	campbell	1	male	166.600	1	37.540	38.011	28.042	3.766	4.128	4.473
3	42.000	whittinger	1	male	183.200	1	50.557	45.633	40.669	3.770	4.008	4.047
4	50.000	sarbo	1	male	176.400	1	37.827	43.803	41.946	4.035	4.026	4.219
5	53.000	germaine	1	male	171.500	1	30.409	36.944	39.901	3.307	3.275	3.648
6	48.000	muldowney	1	male	161.700	1	29.314	34.780	29.286	3.596	3.574	4.070
7	58.000	ochosky	1	male	161.700	1	33.855	28.000	30.083	2.630	3.154	3.546
8	29.000	rose	1	male	166.600	1	47.850	55.500	61.560	4.666	4.668	4.752
9	64.000	sedgrove	1	male	164.150	1	40.902	44.887	43.623	2.702	2.852	3.221
10	41.000	smith	1	male	186.600	1	26.830	23.462	22.565	4.735	4.364	4.897
11	44.000	mcfarland	1	female	161.700	2	40.880	41.394	30.667	2.566	2.093	2.306
12	27.000	viger	1	female	159.250	2	44.044	45.703	49.496	2.555	2.596	2.663
13	23.000	livingstone	1	female	161.700	2	55.870	45.488	56.732	3.159	3.394	3.166
14	23.000	leinhart	1	female	156.800	2	37.965	33.110	33.467	3.231	3.169	2.803
15	23.000	kellal	1	female	161.700	2	36.772	36.073	27.444	3.173	3.079	3.570
16	43.000	hepchin	1	female	151.200	2	46.784	41.696	44.741	2.704	2.733	2.380
17	23.000	fedyma	1	female	154.300	2	38.621	40.719	49.786	2.714	2.767	2.892
18	40.000	gale	2	male	188.300	1	32.242	28.449	28.353	3.553	4.106	3.958
19	41.000	mcginnis	2	male	182.000	1	32.401	28.463	35.183	2.587	2.815	3.273
20	49.000	marsland	2	male	177.600	1	37.924	34.301	36.646	2.731	2.943	3.150
21	24.000	cooper	2	male	182.500	1	33.782	42.890	36.771	3.396	3.883	3.890
22	33.000	dodic	2	male	190.500	1	29.379	24.901	34.103	3.531	4.013	4.048
23	21.000	fisher	2	male	177.500	1	29.761	40.836	39.663	3.184	4.055	4.254
24	30.000	graham	2	male	187.500	1	39.896	40.276	36.477	3.596	4.567	4.479
25	28.000	hanasyck	2	male	173.700	1	36.707	40.419	39.215	3.410	3.966	3.979
26	33.000	mcClure	2	male	162.500	1	41.712	30.130	39.614	2.783	2.906	3.178
27	25.000	kirby	2	male	181.000	1	34.856	41.043	47.662	3.515	4.151	4.359
28	21.000	wall	2	male	186.500	1	36.567	44.177	52.909	3.167	4.170	4.153
29	42.000	Barton	2	male	185.500	1	37.877	36.807	33.553	3.240	3.880	3.420
30	28.000	chalborn	2	male	180.200	1	36.340	37.325	36.649	3.243	4.152	3.873
31	45.000	schultz	2	male	174.700	1	43.649	43.006	43.161	3.459	3.420	3.534
32	32.000	vera	2	female	169.400	2	35.236	23.333	28.059	2.181	2.464	2.622
33	28.000	shaver	2	female	171.100	2	36.636	38.936	19.880	2.274	2.474	2.831
34	27.000	reimer	2	female	167.400	2	23.257	27.554	39.186	2.667	3.354	3.120
35	28.000	paradson	2	female	160.800	2	31.366	39.965	30.220	2.311	2.746	2.909
36	4[illegible].000	mcginnis	2	female	165.900	2	26.239	26.185	29.420	2.099	2.491	2.645
37	2[illegible].000	murphy	2	female	173.100	2	48.244	36.104	39.266	2.165	2.458	2.326
38	24.000	day	2	female	160.020	2	29.639	17.676	26.881	1.965	2.232	2.403
39	41.000	blesser	2	female	154.000	2	35.182	45.639	44.168	1.778	2.005	2.466
40	22.000	barry	2	female	182.300	2	35.215	30.727	34.688	2.067	2.792	2.787
41	29.000	askin	2	female	164.700	2	36.882	34.213	34.722	2.576	2.725	3.493
42	33.000	grant	2	female	166.000	2	41.820	40.642	36.157	1.956	2.222	2.332
43	26.000	hiron	2	female	155.500	2	28.711	35.213	31.981	2.118	2.487	2.345
44	28.000	mcclure	2	female	162.000	2	21.021	21.340	25.500	1.462	1.780	1.714
45	22.000	woudstra	3	male	169.050	1	52.123	44.378	41.320	3.161	3.340	3.340
46	32.000	welycoshy	3	male	164.150	1	36.173	34.734	40.267	3.009	3.009	3.261
47	23.000	wreimer	3	male	170.275	1	28.647	37.236	40.877	3.637	4.011	4.218
48	29.000	ingham	3	male	171.500	1	29.302	29.205	33.637	3.662	3.749	3.888
49	31.000	albright	3	male	171.500	1	34.784	33.317	27.665	2.925	3.226	3.053
50	22.000	mcdonald	3	male	169.100	1	.	.	.	.	.	.
51	23.000	hornford	3	male	170.250	1	31.997	29.448	34.427	3.170	3.176	3.183
52	23.000	kennedy	3	male	173.950	1	.	.	.	.	.	.
53	36.000	semasky...	3	female	156.830	2	.	.	.	.	.	.
54	21.000	schaluk	3	female	150.250	2	42.477	61.766	45.074	1.865	2.233	1.827
55	21.000	searle	3	female	161.700	2	43.039	50.217	41.832	1.963	2.017	2.193
56	34.000	samuels	3	female	149.450	2	46.045	46.583	70.179	2.381	2.633	2.505
57	26.000	richardson	3	female	160.000	2	24.108	26.573	32.934	1.965	2.001	2.145
58	30.000	olson	3	female	156.800	2	.	.	.	.	.	.
59	25.000	mish	3	female	159.400	2	25.993	29.696	27.721	1.999	1.983	2.041
60	24.000	harbin	3	female	157.800	2	40.702	29.603	28.778	1.918	1.793	1.830
61	24.000	gaunt	3	female	155.300	2	25.400	21.160	22.928	1.864	1.765	1.873
62	23.000	hagen	3	female	164.150	2	35.169	30.136	26.854	2.319	2.263	2.523
63	30.000	cooper	3	female	166.000	2	.	.	.	.	.	.
64	.		.		.	.	.	.	.	.	.	.
65	.		.		.	.	.	.	.	.	.	.

	kcals/lbm pre	kcals/lbm mid	kcals/lbmpost	fat/kg pre	fat kg mid	fat kg post	prot kg pre	prot kg mid
1	36.171	32.510	30.086	1.364	.717	.914	1.286	1.167
2	42.589	44.110	30.546	.941	.822	.876	1.302	1.351
3	69.463	51.178	56.063	2.200	1.889	2.132	2.102	1.983
4	46.414	56.338	53.486	1.574	1.905	1.606	1.421	1.380
5	33.752	40.609	42.833	1.258	1.392	1.609	1.179	1.472
6	36.319	45.969	39.412	.843	1.129	.987	1.186	1.290
7	43.824	32.106	35.186	1.706	1.164	1.500	1.201	1.377
8	53.439	66.109	64.676	1.83	1.867	2.400	1.567	1.960
9	47.981	53.161	52.074	1.459	1.290	1.541	1.492	1.145
10	27.942	25.388	24.246	.986	.804	.733	1.127	1.080
11	56.567	63.567	40.292	1.109	1.505	.907	1.592	1.541
12	54.112	56.867	62.293	1.753	2.186	1.841	1.614	1.680
13	68.753	56.914	72.000	1.874	1.262	1.362	2.256	1.683
14	46.398	40.913	40.624	1.490	1.168	1.099	1.196	1.254
15	41.486	40.958	31.826	1.063	1.174	.672	1.382	1.229
16	60.214	49.355	53.782	1.743	1.342	1.573	2.116	1.667
17	45.025	47.102	58.250	1.164	1.163	1.581	1.724	1.607
18	39.544	32.489	29.973	.686	.477	.326	1.319	.931
19	41.740	34.794	47.400	1.432	1.347	1.606	1.241	.966
20	49.541	44.306	46.262	1.206	1.091	1.071	1.084	1.352
21	39.356	48.987	43.630	2.082	1.771	1.402	1.728	1.501
22	33.627	28.898	38.185	1.071	.978	1.207	1.167	1.077
23	36.331	48.421	46.820	1.449	1.739	1.464	1.612	1.813
24	46.277	48.990	42.686	1.497	1.325	1.114	1.645	1.501
25	46.711	47.878	45.243	1.628	1.937	1.332	1.211	1.178
26	49.104	46.023	49.280	1.664	1.079	1.158	1.743	1.675
27	39.863	46.640	53.714	1.127	1.157	1.479	1.479	1.500
28	43.636	52.190	64.966	1.217	1.616	2.053	1.413	1.369
29	46.544	46.656	41.329	1.128	1.404	1.092	1.798	1.106
30	43.270	44.041	42.169	1.600	1.166	1.099	1.429	1.263
31	53.590	49.826	51.625	1.522	1.486	1.462	1.721	1.949
32	48.599	31.111	35.931	1.289	.846	1.079	1.073	.799
33	55.153	53.140	27.221	1.979	1.529	.695	1.083	1.170
34	32.931	38.269	51.663	.700	1.046	1.615	1.035	1.262
35	41.143	50.728	46.830	.974	1.374	1.294	1.368	1.304
36	37.467	36.104	38.382	.917	1.224	.741	1.147	.785
37	61.175	46.046	47.838	1.512	1.477	1.433	1.089	1.299
38	45.409	24.206	36.792	1.036	.487	.784	1.250	.767
39	52.534	58.221	61.691	1.299	1.552	1.983	1.277	2.122
40	48.393	40.723	46.552	1.092	.909	.997	1.092	1.288
41	47.678	45.956	•	1.107	1.134	1.113	1.471	1.506
42	62.126	59.375	50.403	1.749	1.765	1.549	1.431	1.462
43	39.426	47.626	39.880	1.132	1.613	1.136	1.098	1.330
44	28.636	29.827	35.376	.880	.577	.771	1.149	.948
45	57.156	47.147	45.169	1.604	1.606	1.510	1.714	1.496
46	44.261	42.522	49.861	1.453	1.540	1.810	1.252	1.252
47	32.868	43.007	46.846	1.479	1.399	1.358	.940	1.032
48	35.862	35.186	38.913	1.552	1.398	1.374	1.519	1.523
49	41.331	39.609	33.012	1.392	1.234	1.063	1.312	1.266
50	•	•	•	•	•	•	•	•
51	37.552	36.335	42.662	.947	1.030	.813	1.272	1.239
52	•	•	•	•	•	•	•	•
53	•	•	•	•	•	•	•	•
54	60.969	67.910	67.876	1.509	2.751	2.048	1.155	1.580
55	51.769	62.729	51.448	1.746	2.034	1.794	1.808	1.771
56	55.246	60.600	63.523	2.272	2.046	3.147	2.028	2.196
57	32.120	36.102	44.083	1.002	1.167	.985	.923	1.129
58	•	•	•	•	•	•	•	•
59	37.247	42.436	40.189	1.051	1.380	1.025	1.248	1.153
60	55.859	40.761	39.974	2.334	.983	.902	1.537	1.040
61	32.353	26.909	29.363	.909	.773	.884	.945	.755
62	48.032	42.033	36.299	1.339	1.237	.986	1.169	1.153
63	•	•	•	•	•	•	•	•
64	•	•	•	•	•	•	•	•
65	•	•	•	•	•	•	•	•

	fat lbm post	prot lbm pre	prot lbm mid	prot lbm post	cho lbm pre	cho lbm mid	cho lbm post	deficit/kg pre
1	1.029	1.479	1.373	1.181	3.949	4.804	3.733	-15.008
2	.736	1.477	1.528	1.288	6.103	6.220	4.188	1.166
3	2.407	2.452	2.224	2.052	8.276	4.804	5.149	3.585
4	2.060	1.818	1.744	2.158	4.403	5.694	5.701	-16.357
5	1.731	1.308	1.618	1.519	4.049	5.311	5.654	-6.912
6	1.328	1.488	1.705	1.518	4.336	4.535	4.573	-13.541
7	1.754	1.554	1.700	1.657	4.744	3.482	3.646	-6.739
8	2.522	1.757	2.112	2.224	6.318	7.238	6.865	-2.058
9	1.840	1.750	1.356	1.409	6.298	8.346	8.043	-13.428
10	.788	1.174	1.166	1.041	3.619	3.362	3.390	-16.903
11	1.192	2.214	1.996	1.533	8.383	7.221	5.523	1.962
12	2.317	1.982	2.054	2.171	7.220	6.257	8.610	2.769
13	1.728	2.776	2.105	2.593	8.682	8.828	9.111	8.927
14	1.335	1.462	1.550	1.615	5.972	5.425	5.561	-2.073
15	.779	1.537	1.396	1.103	6.211	5.562	5.084	-7.285
16	1.891	2.724	2.233	2.487	7.770	6.799	6.969	7.488
17	1.850	2.010	1.859	1.925	6.281	6.848	8.250	-5.306
18	.371	1.818	1.063	1.223	6.103	5.995	5.975	-.259
19	2.163	1.599	1.270	1.594	3.972	3.202	3.985	-.896
20	1.352	1.416	1.746	2.008	6.821	6.190	6.441	2.232
21	1.664	2.013	1.715	1.647	7.112	5.987	5.361	-3.120
22	1.351	1.336	1.240	1.377	3.002	3.024	4.399	4.926
23	1.728	1.968	2.149	1.865	3.275	4.254	4.758	-3.404
24	1.303	1.748	1.826	1.463	5.648	6.242	6.064	8.835
25	1.537	1.537	1.395	1.942	5.372	5.426	5.874	3.917
26	1.440	2.052	1.970	2.600	5.037	5.739	5.980	6.819
27	1.667	1.683	1.705	1.794	4.535	5.633	6.667	-.858
28	2.517	1.686	1.618	1.784	6.072	7.337	8.870	.007
29	1.345	2.301	1.371	1.507	6.478	6.487	5.381	5.125
30	1.265	1.701	1.490	1.506	3.590	4.941	4.849	3.770
31	1.749	2.112	2.259	2.332	5.840	6.466	6.714	9.651
32	1.381	1.480	1.053	1.125	5.781	4.172	4.045	3.594
33	1.225	1.546	1.597	1.170	5.916	7.405	3.053	7.620
34	2.130	1.466	1.752	1.823	4.501	4.829	6.430	-8.143
35	1.611	1.794	1.656	1.943	5.336	6.358	6.225	-1.636
36	.966	1.638	1.082	2.248	2.860	3.139	2.143	-4.528
37	1.746	1.381	1.656	1.622	10.000	5.694	6.424	12.348
38	1.053	1.902	1.077	1.353	5.815	3.282	4.160	-3.739
39	2.750	1.907	2.707	2.148	6.158	5.815	6.260	2.348
40	1.339	1.501	1.707	1.785	6.469	5.803	6.227	.437
41	1.451	1.964	2.023	2.066	5.862	5.227	5.253	1.121
42	2.159	2.126	2.135	1.821	7.428	4.245	5.852	6.260
43	1.415	1.507	1.799	2.086	4.866	5.348	4.173	-4.219
44	1.069	1.565	1.326	1.676	3.188	4.006	4.220	-15.234
45	1.651	1.879	1.589	1.460	8.862	6.525	6.083	14.959
46	2.365	1.532	1.532	1.443	4.032	4.140	4.643	5.424
47	1.556	1.079	1.192	1.309	3.500	5.589	6.639	.021
48	1.590	1.850	1.835	1.723	2.754	3.163	4.221	1.695
49	1.268	1.559	1.505	1.590	5.456	4.895	3.859	1.157
50	.	.	.	.	.	.	.	.
51	1.007	1.493	1.529	2.302	5.139	4.825	6.133	-1.111
52	.	.	.	.	.	.	.	.
53	.	.	.	.	.	.	.	.
54	3.903	1.658	2.249	2.266	8.476	11.481	11.079	8.402
55	2.207	1.934	2.212	1.747	6.392	7.529	6.253	7.925
56	3.746	2.434	2.675	3.556	4.989	5.800	8.608	12.284
57	1.318	1.230	1.532	1.266	3.613	4.059	7.132	-8.407
58	.	.	.	.	.	.	.	.
59	1.486	1.788	1.647	1.344	4.424	4.826	5.684	-3.662
60	1.253	2.109	1.432	1.482	4.896	5.964	5.405	6.901
61	1.132	1.204	.960	1.038	4.447	2.646	3.774	-5.811
62	1.333	1.597	1.608	1.724	5.949	5.059	4.391	2.271
63	.	.	.	.	.	.	.	.
64	.	.	.	.	.	.	.	.
65	.	.	.	.	.	.	.	.

	prot kg post	cho kg pre	cho kg mid	cho kg post	cal lbm pre	cal lbm mid	cal lbm post	fat lbm pre	fat lbm mid
1	1.049	3.435	4.083	3.316	38.171	32.510	30.095	1.556	.843
2	1.183	5.384	5.501	3.846	42.559	44.110	30.546	1.066	.929
3	1.818	7.094	4.283	4.562	69.483	51.178	58.063	2.567	2.112
4	1.883	3.440	4.507	4.471	48.414	55.338	53.489	2.014	2.278
5	1.412	3.648	4.832	5.256	33.752	40.608	42.933	1.398	1.530
6	1.128	3.500	3.431	3.399	36.319	45.969	39.412	1.044	1.492
7	1.417	3.865	2.820	3.117	43.824	32.105	35.186	2.209	1.437
8	2.117	5.833	6.663	6.533	53.439	60.108	64.676	2.056	1.805
9	1.180	5.361	7.048	6.738	47.981	53.151	52.074	1.712	1.528
10	.960	3.475	3.109	3.154	27.942	25.356	24.248	1.036	.869
11	1.167	6.029	5.576	4.204	56.567	53.587	40.292	1.542	1.948
12	1.725	5.876	5.117	6.841	54.112	55.887	62.293	2.154	2.675
13	2.043	7.055	7.055	7.179	68.753	56.914	72.000	2.306	1.579
14	1.330	4.885	4.390	4.580	46.398	40.913	40.624	1.822	1.444
15	.951	5.504	4.899	4.384	41.495	40.958	31.826	1.200	1.333
16	2.069	6.037	5.744	5.797	60.214	49.355	53.782	2.243	1.588
17	1.645	5.388	5.920	7.051	45.025	47.102	58.250	1.357	1.345
18	1.075	4.976	5.251	5.254	39.544	32.480	29.973	.853	.545
19	1.163	3.083	2.435	2.958	41.740	34.794	47.400	1.845	1.771
20	1.591	5.221	4.793	5.102	49.541	44.308	46.262	1.576	1.409
21	1.388	6.105	5.241	4.518	39.358	46.997	43.630	2.426	2.023
22	1.230	2.623	2.626	3.920	33.627	28.669	38.185	1.225	1.126
23	1.580	2.683	3.588	4.028	36.331	48.421	46.820	1.768	2.061
24	1.250	4.867	5.132	5.182	48.277	48.980	42.686	1.811	1.611
25	1.683	4.232	4.581	5.091	46.711	47.878	45.243	2.066	2.295
26	2.090	4.279	4.879	4.807	40.104	46.023	40.280	1.959	1.269
27	1.592	3.966	4.957	5.915	39.863	46.640	53.714	1.282	1.315
28	1.439	5.091	6.210	7.236	43.636	52.190	64.966	1.451	1.912
29	1.224	5.063	5.230	4.368	48.544	45.655	41.329	1.443	1.741
30	1.309	3.016	4.188	4.216	43.270	44.041	42.169	1.904	1.401
31	1.950	4.758	5.590	5.613	53.580	49.828	51.625	1.868	1.724
32	.878	4.192	3.129	3.159	48.599	31.111	35.891	1.736	1.131
33	.854	4.144	5.428	2.230	56.153	53.140	27.221	2.824	2.087
34	1.231	3.242	3.477	4.877	32.931	38.269	51.863	.991	1.453
35	1.560	4.068	5.009	5.000	41.143	50.728	48.630	1.278	1.744
36	1.723	2.003	2.276	1.843	37.467	36.104	38.382	1.310	1.688
37	1.331	7.886	4.484	5.273	61.175	46.046	47.838	1.918	1.884
38	.982	3.821	2.397	3.018	45.408	24.205	36.792	1.576	.667
39	1.533	4.124	4.588	4.467	52.534	58.221	61.801	1.880	1.960
40	1.329	4.708	4.379	4.637	48.393	40.723	46.552	1.501	1.208
41	1.585	4.412	3.892	4.030	47.676	45.955	45.253	1.471	1.523
42	1.306	5.000	2.906	4.198	62.126	50.375	50.403	2.598	2.578
43	1.673	3.397	3.964	3.346	39.426	47.626	39.880	1.555	2.182
44	1.208	2.340	2.886	3.042	28.638	29.827	35.376	.890	.807
45	1.338	8.082	6.142	5.584	57.155	47.147	45.169	1.750	1.707
46	1.166	3.295	3.381	3.750	44.261	42.522	49.861	1.778	1.885
47	1.142	3.050	4.839	5.793	32.868	43.007	46.846	1.697	1.616
48	1.490	2.251	2.625	3.649	35.862	35.186	38.913	1.900	1.684
49	1.332	4.592	4.119	3.234	41.331	39.600	33.012	1.654	1.467
50	.	.	.	.	.	.	.	.	.
51	1.858	4.379	3.910	4.949	37.552	38.335	42.662	1.111	1.271
52	.	.	.	.	.	.	.	.	.
53	.	.	.	.	.	.	.	.	.
54	1.162	5.903	6.067	5.663	60.969	67.910	67.676	2.166	3.915
55	1.421	5.314	6.028	5.084	51.769	62.729	51.448	2.099	2.541
56	2.986	4.158	4.696	7.231	55.246	60.000	63.523	2.728	2.525
57	.946	2.711	2.988	5.328	32.120	38.102	44.083	1.335	1.586
58	.	.	.	.	.	.	.	.	.
59	.927	3.087	3.377	3.921	37.247	42.436	40.189	1.506	1.972
60	1.053	3.567	4.329	3.891	55.859	40.781	39.974	3.203	1.354
61	.810	3.491	2.081	2.947	32.353	26.909	29.363	1.158	.984
62	1.278	4.356	3.627	3.246	48.032	42.033	36.299	1.829	1.726
63	.	.	.	.	.	.	.	.	.
64	.	.	.	.	.	.	.	.	.
65	.	.	.	.	.	.	.	.	.

	deficit/kg post
1	-21.301
2	-6.508
3	-2.407
4	.969
5	-8.135
6	-6.423
7	-13.319
8	12.820
9	-5.264
10	-22.774
11	-10.465
12	7.975
13	4.932
14	-8.137
15	-17.349
16	1.741
17	6.006
18	-4.999
19	-4.931
20	-3.235
21	-3.601
22	-5.298
23	.961
24	-1.587
25	-1.324
26	-1.513
27	5.737
28	9.441
29	-5.847
30	-4.847
31	.363
32	-7.844
33	-13.961
34	.149
35	.982
36	-6.652
37	.643
38	-11.438
39	3.884
40	-2.467
41	-5.626
42	-3.284
43	-8.529
44	-15.325
45	5.240
46	9.306
47	10.465
48	4.823
49	-3.642
50	•
51	1.478
52	•
53	•
54	11.993
55	7.984
56	34.247
57	-.131
58	•
59	-2.020
60	-3.741
61	-3.565
62	-5.313
63	•
64	•
65	•

Appendix 4. Dietary Intake in]0 HMR.

	DIARY NUMBER	BARTON	SCHULTZ	MCCLURE	HANASYCK
1	1	3068.000	3038.000	3337.000	3346.000
2	2	3068.000	3038.000	4274.000	2304.000
3	3	3231.000	2465.000	1034.000	2871.000
4	4	3005.000	2375.000	2310.000	2245.000
5	5	2755.000	2146.000	2092.000	2603.000
6	6	2616.000	2326.000	2140.000	3284.000
7	7	2561.000	2932.000	2280.000	2928.000
8	8	2441.000	2846.000	2969.000	3270.000
9	9	3189.000	2258.000	3005.000	2717.000
10	10	2774.000	2363.000	2871.000	2605.000
11	11	2817.000	3422.000	2455.000	3884.000
12	12	2356.000	3156.000	2552.000	3855.000
13	13	2443.000	2601.000	2454.000	1736.000
14	14	2656.000	3241.000	2139.000	2228.000

	GRAHAM	DODIC	CHALBORN	FISHER	WALL	KIRBY
1	4164.000	2272.000	2498.000	3581.000	2591.000	2463.000
2	3510.000	2272.000	3455.000	3289.000	2641.000	2502.000
3	3283.000	3222.000	2842.000	2254.000	2790.000	2605.000
4	3271.000	2892.000	2707.000	2472.000	2647.000	2688.000
5	2966.000	2837.000	2422.000	2238.000	3207.000	3010.000
6	3181.000	3244.000	3118.000	2686.000	3557.000	3043.000
7	4123.000	2171.000	2853.000	3253.000	2830.000	2706.000
8	4049.000	2361.000	2529.000	3310.000	3531.000	3477.000
9	3684.000	2265.000	3022.000	3313.000	2674.000	2207.000
10	3945.000	2464.000	3147.000	4142.000	3430.000	2593.000
11	3402.000	2249.000	2802.000	4084.000	3500.000	3712.000
12	3030.000	3149.000	2855.000	3363.000	4289.000	2684.000
13	4526.000	3303.000	2744.000	2813.000	3097.000	4084.000
14	3711.000	2631.000	2535.000	3310.000	2708.000	3874.000

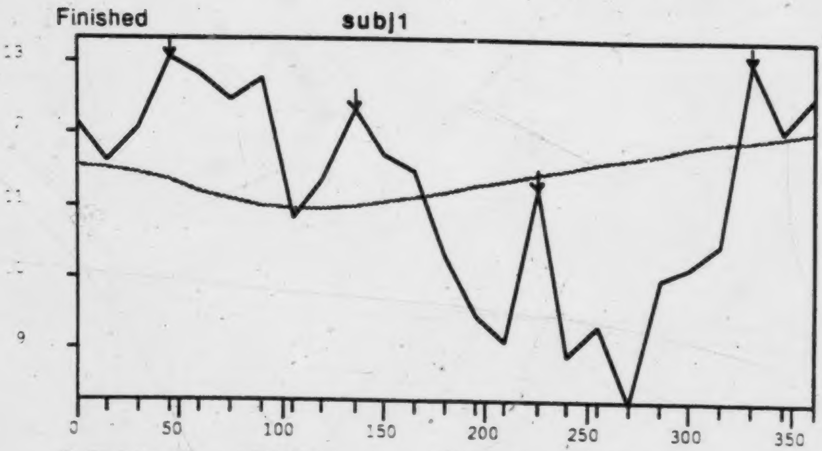
	bartondis	schultzdis	mccluredis	hanasyckdis	grahamdis
1	0	0	0	0	0
2	0	0	0	0	0
3	7.200	19.200	18.080	8.100	10.400
4	8.000	24.000	17.200	12.100	10.750
5	11.000	25.160	26.900	10.300	14.300
6	18.200	33.000	25.500	13.500	16.300
7	26.800	28.800	38.680	21.900	12.100
8	27.200	35.200	35.800	20.400	28.500
9	33.800	43.200	42.000	16.450	22.250
10	33.250	48.000	50.800	29.600	33.500
11	34.900	48.000	56.000	39.200	36.100
12	40.800	48.000	60.080	34.000	33.150
13	36.800	48.000	60.600	36.400	35.750
14	42.000	48.000	54.800	39.200	23.500

	dodicdis	chalborndis	fisherdis	walldis	kirbydis
1	0	0	0	0	0
2	0	0	0	0	0
3	8.400	9.200	7.200	7.600	7.600
4	8.000	9.200	9.800	9.280	9.600
5	11.600	12.400	13.800	11.120	11.800
6	12.600	14.600	13.300	14.560	12.250
7	15.000	17.000	16.400	15.200	18.200
8	17.500	17.650	22.400	20.800	22.300
9	22.500	5.600	12.400	23.360	18.150
10	20.800	18.400	28.800	18.000	22.400
11	35.000	30.150	12.800	28.400	38.550
12	32.650	23.500	12.800	28.800	32.000
13	41.800	41.000	24.400	27.600	40.000
14	50.800	48.500	24.600	43.200	39.600

APPENDIX 5A.

LUTEINIZING HORMONE DATA
SIX HOUR SAMPLING PROFILES: HIGH MILEAGE RUNNERS

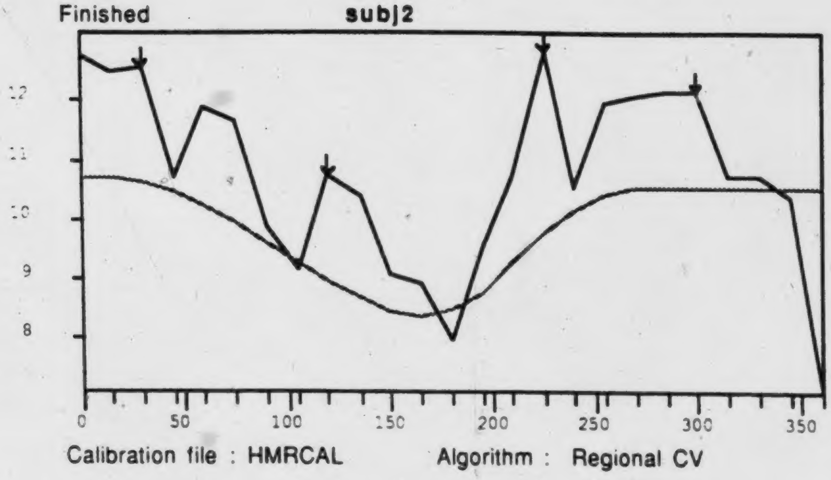
```
Individual ID  subj1
```



Calibration file : HMRCAL   Algorithm : Regional CV

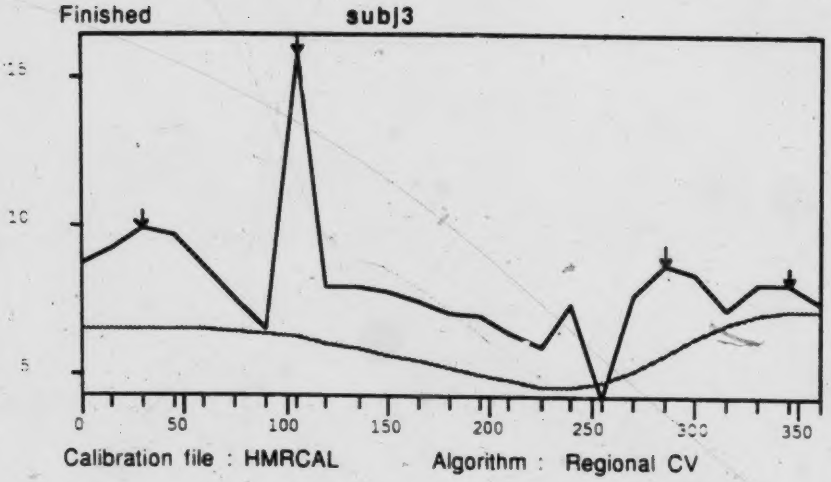
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =   96.57
Between replicates sum of squares =   10.31
F =    9.74 (dF = 24, 25)
Signal to noise ratio  =    2.95

number of peaks              4
mean pulse interval         95.00      8.66 s.d.
mean pulse amplitude         2.46      1.56 s.d.
mean pulse area            -29.23     99.96 s.d.
mean nadir                   9.94      1.53 s.d.
mean measured level         11.18


Time      Amplitude  Nadir  Area
  45.0       1.45    11.60   106.45
 135.0       1.51    10.81   -18.39
 225.0       2.12     9.11  -121.37
 330.0       4.77     8.25   -83.62
```

Individual ID subj2



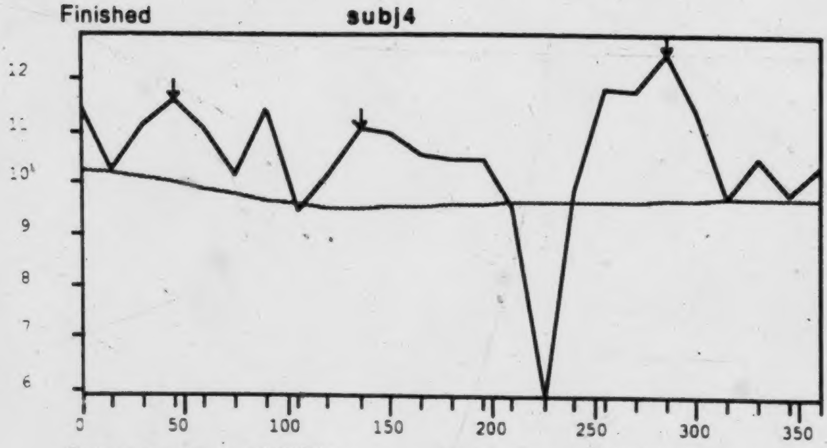
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  113.35
Between replicates sum of squares =   20.20
F =    5.84 (dF = 24, 25)
Signal to noise ratio  =    2.20

number of peaks             4
mean pulse interval        90.00     15.00 s.d.
mean pulse amplitude        2.04      2.01 s.d.
mean pulse area            79.45     15.46 s.d.
mean nadir                 10.03      1.93 s.d.
mean measured level        10.75


Time    Amplitude  Nadir  Area
  30.0      0.09   12.47   101.01
 120.0      1.57    9.17    64.46
 225.0      4.87    7.95    77.93
 300.0      1.63   10.52    74.39
```

```
Individual ID  subj3
```



```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  195.32
Between replicates sum of squares =   18.32
F =   11.10 (dF = 24, 25)
Signal to noise ratio  =    3.17

number of peaks             4
mean pulse interval         105.00     65.38 s.d.
mean pulse amplitude          3.97      3.92 s.d.
mean pulse area             190.04    159.20 s.d.
mean nadir                    6.67      1.89 s.d.
mean measured level           8.04


Time    Amplitude  Nadir  Area
  30.0     1.15     8.77   208.27
 105.0     9.30     6.50   403.81
 285.0     4.53     4.21   113.44
 345.0     0.89     7.22    34.65
```

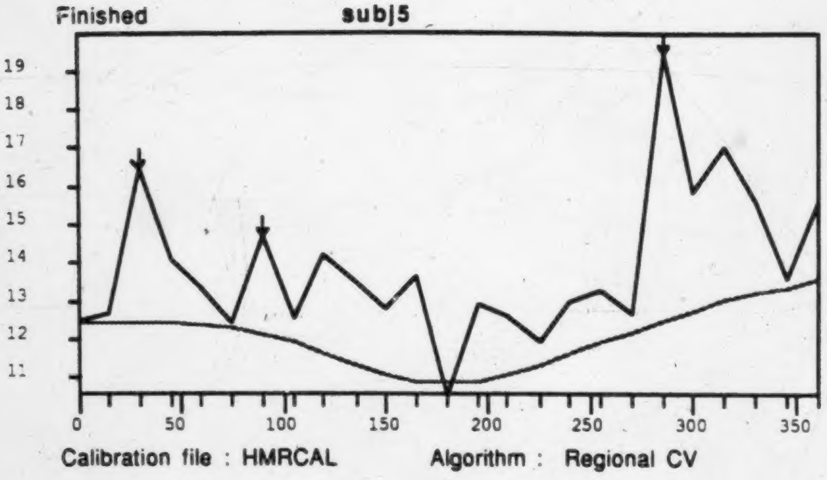
Individual ID subj4



Calibration file : HMRCAL   Algorithm : Regional CV

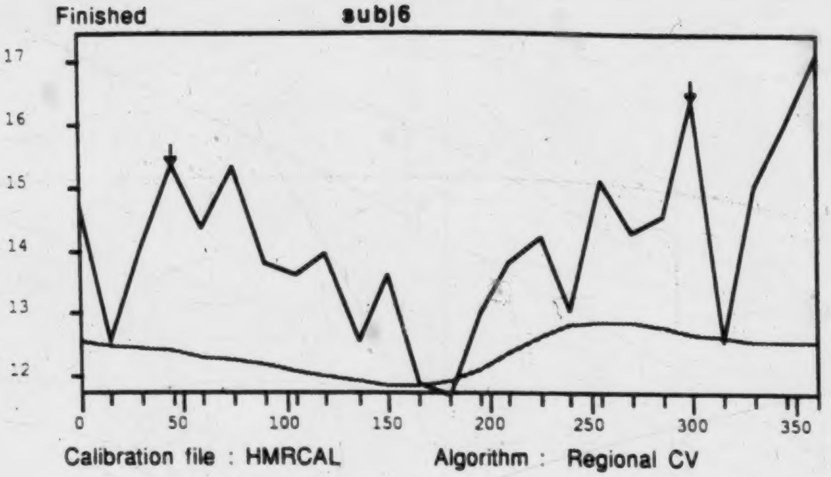
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =   76.04
Between replicates sum of squares =   18.13
F =    4.36 (dF = 24, 25)
Signal to noise ratio  =    1.83

number of peaks           3
mean pulse interval     120.00     42.42 s.d.
mean pulse amplitude      3.20      3.01 s.d.
mean pulse area          86.74     21.06 s.d.
mean nadir                8.52      2.36 s.d.
mean measured level      10.54


Time     Amplitude  Nadir   Area
  45.0      1.35   10.26     89.89
 135.0      1.57    9.48     64.28
 285.0      6.68    5.83    106.04
```

Individual ID subj5



```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  176.64
Between replicates sum of squares =   32.46
F =     5.66 (dF = 24, 25)
Signal to noise ratio  =     2.16

number of peaks           3
mean pulse interval       127.50     95.45 s.d.
mean pulse amplitude        4.60      2.68 s.d.
mean pulse area           197.99     98.53 s.d.
mean nadir                 12.27      0.30 s.d.
mean measured level        13.87


Time     Amplitude  Nadir  Area
  30.0      4.00   12.48    105.91
  90.0      2.27   12.42    186.15
 285.0      7.54   11.92    301.90
```

ID subj6

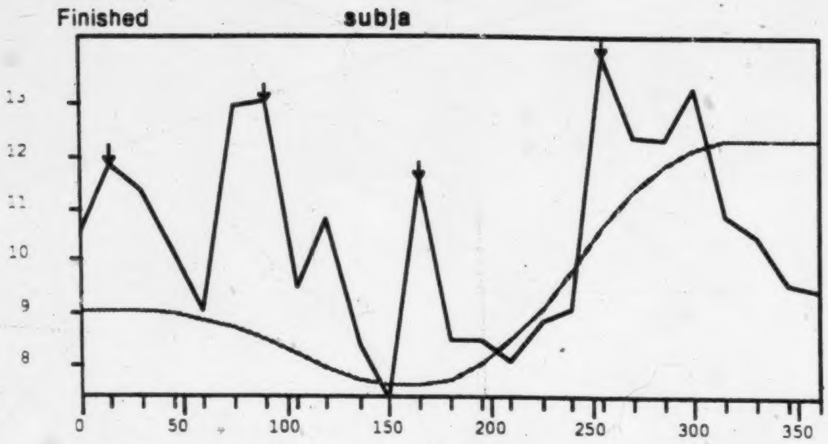


```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =   89.30
Between replicates sum of squares =   34.45
F =    2.70 (dF = 24, 25)
Signal to noise ratio  =    1.30

number of peaks          2
mean pulse interval    255.00
mean pulse amplitude     3.08      0.39 s.d.
mean pulse area        197.21     82.81 s.d.
mean nadir              12.81      0.34 s.d.
mean measured level     14.11


Time    Amplitude  Nadir  Area
  45.0      2.80   12.57    255.77
 300.0      3.36   13.06    138.65
```

APPENDIX 5B.

LUTEINIZING HORMONE DATA.
SIX HOUR SMPLING PROFILES: TRAINEE PRE AND POST

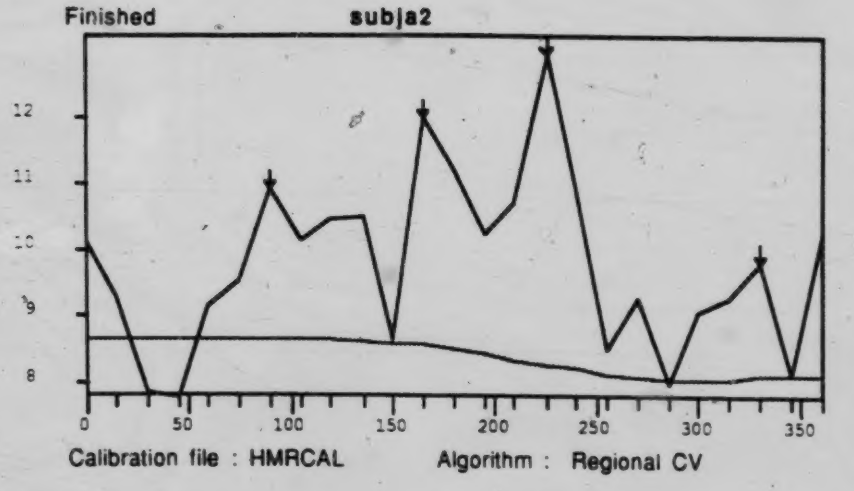
Individual ID subja



Calibration file : HMRCAL Algorithm : Regional CV

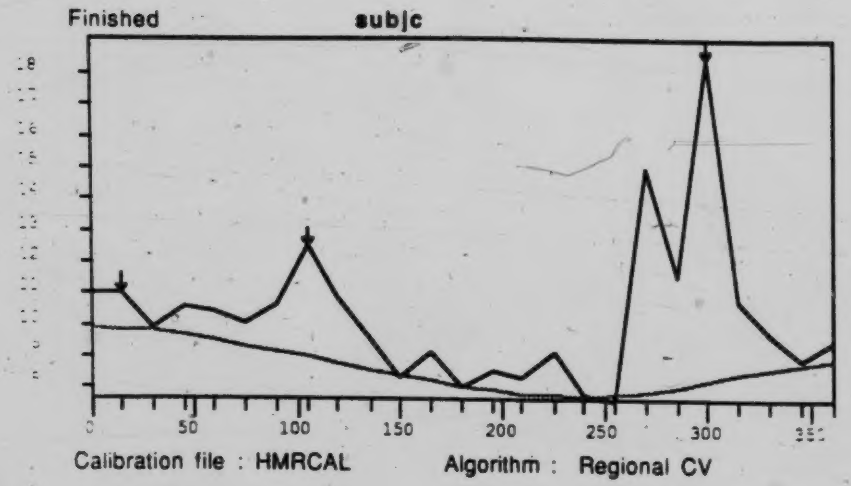
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  161.50
Between replicates sum of squares =   13.38
F =   12.56 (dF = 24, 25)
Signal to noise ratio  =    3.40

number of peaks            4
mean pulse interval         80.00       8.66 s.d.
mean pulse amplitude         3.81       1.88 s.d.
mean pulse area             84.63     101.61 s.d.
mean nadir                   8.77       1.38 s.d.
mean measured level         10.47


Time     Amplitude  Nadir   Area
  15.0      1.27    10.56   106.77
  90.0      3.98     9.05   203.39
 165.0      4.16     7.37    71.04
 255.0      5.83     8.10   -42.64
```

Individual ID subja2



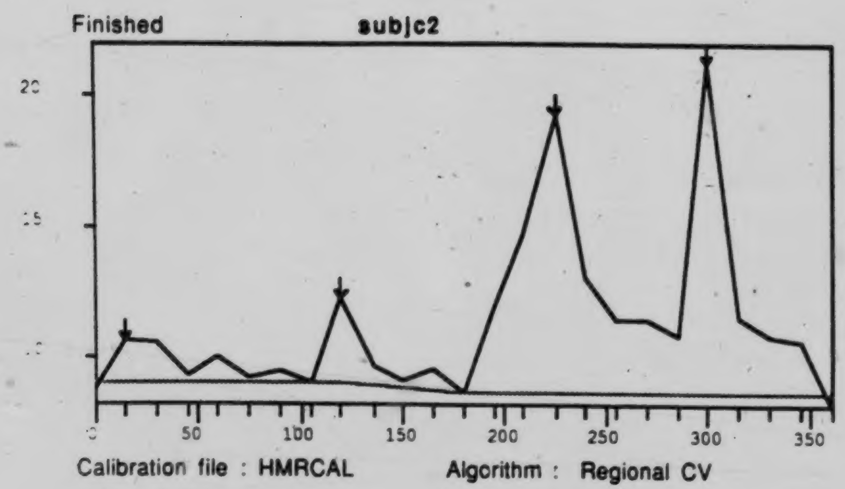
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =   81.19
Between replicates sum of squares =   18.58
F =    4.55 (dF = 24, 25)
Signal to noise ratio  =    1.88

number of peaks             4
mean pulse interval         80.00     22.91 s.d.
mean pulse amplitude         2.76      0.67 s.d.
mean pulse area            119.55     51.06 s.d.
mean nadir                   8.66      1.12 s.d.
mean measured level          9.79


Time    Amplitude  Nadir  Area
  90.0      3.16    7.76   126.48
 165.0      3.35    8.64   107.51
 225.0      2.70   10.25   183.82
 330.0      1.84    7.98    60.39
```

```
Individual ID  subjc
```



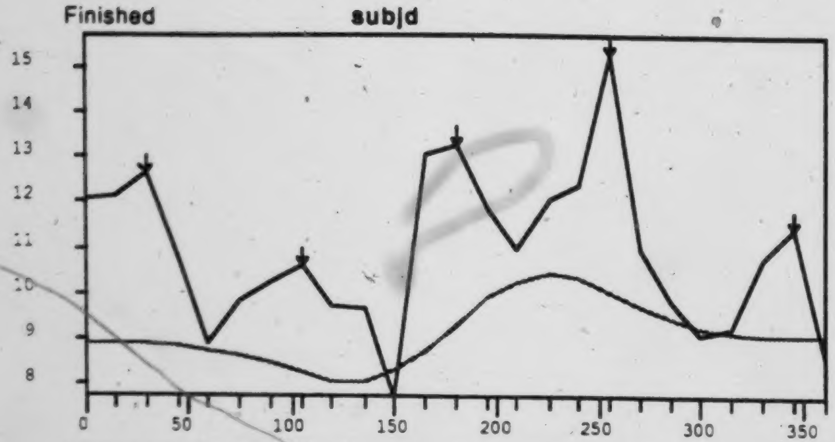
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  268.80
Between replicates sum of squares =   15.68
F =    17.85 (dF = 24, 25)
Signal to noise ratio  =    4.10

number of peaks            3
mean pulse interval        142.50     74.24 s.d.
mean pulse amplitude         4.46      5.76 s.d.
mean pulse area            190.30    168.69 s.d.
mean nadir                   9.57      1.80 s.d.
mean measured level         10.31


Time     Amplitude  Nadir  Area
  15.0      0.00    11.08    29.01
 105.0      2.42    10.08   176.36
 300.0     10.97     7.57   365.53
```

Individual ID subjc2



```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  464.50
Between replicates sum of squares =   26.44
F =   18.29 (dF = 24, 25)
Signal to noise ratio  =    4.15

number of peaks               4
mean pulse interval         95.00      17.32 s.d.
mean pulse amplitude         6.49       4.63 s.d.
mean pulse area            227.81     191.44 s.d.
mean nadir                   9.35       1.01 s.d.
mean measured level         11.25


Time     Amplitude  Nadir  Area
  15.0      1.82     8.81    69.05
 120.0      3.20     9.07    74.74
 225.0     10.58     8.68   463.40
 300.0     10.36    10.85   304.05
```

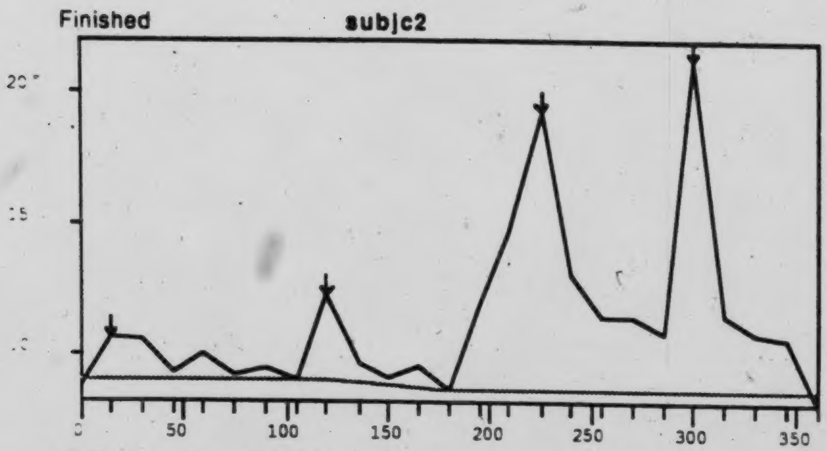
Individual ID subjd



Calibration file : HMRCAL Algorithm : Regional CV

```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  143.48
Between replicates sum of squares =   12.94
F =   11.55 (dF = 24, 25)
Signal to noise ratio  =    3.24

number of peaks            5
mean pulse interval        78.75      7.50 s.d.
mean pulse amplitude        2.90      2.03 s.d.
mean pulse area           131.64     45.13 s.d.
mean nadir                  9.75      1.74 s.d.
mean measured level        10.94


Time     Amplitude  Nadir   Area
  30.0      0.55    12.08   161.62
 105.0      1.71     8.87   123.39
 180.0      5.59     7.71   154.86
 255.0      4.33    10.96   162.22
 345.0      2.33     9.12    56.10
```

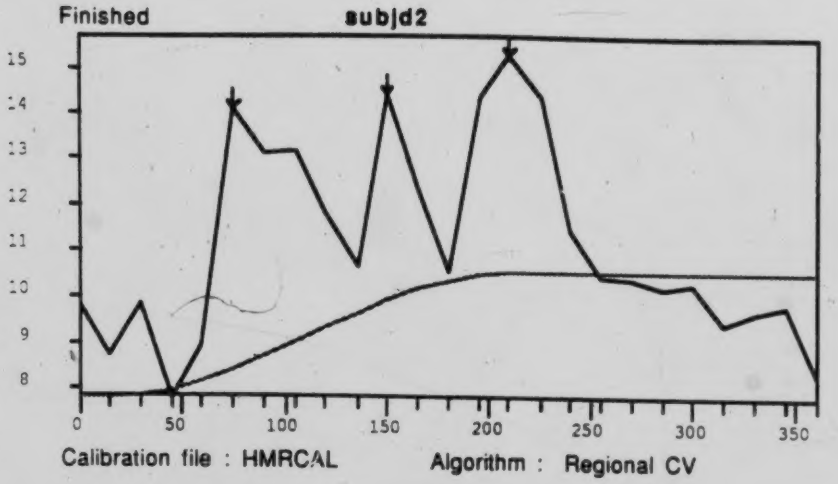
Individual ID subjc2



Calibration file : HMRCAL Algorithm : Regional CV

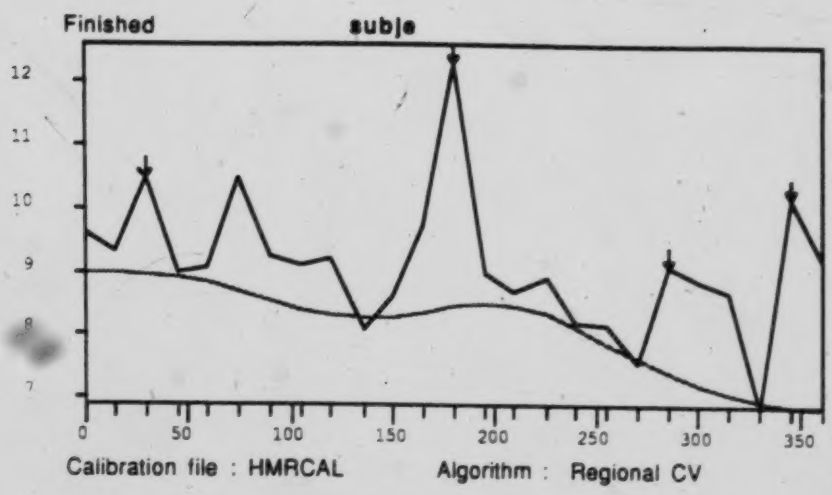
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  464.50
Between replicates sum of squares =   26.44
F =   18.29 (dF = 24, 25)
Signal to noise ratio  =    4.15

number of peaks             4
mean pulse interval        95.00      17.32 s.d.
mean pulse amplitude        6.49       4.63 s.d.
mean pulse area           227.81     191.44 s.d.
mean nadir                  9.35       1.01 s.d.
mean measured level        11.25


Time     Amplitude  Nadir  Area
  15.0      1.82     8.81     69.05
 120.0      3.20     9.07     74.74
 225.0     10.58     8.68    463.40
 300.0     10.36    10.85    304.05
```

Individual ID subjd2



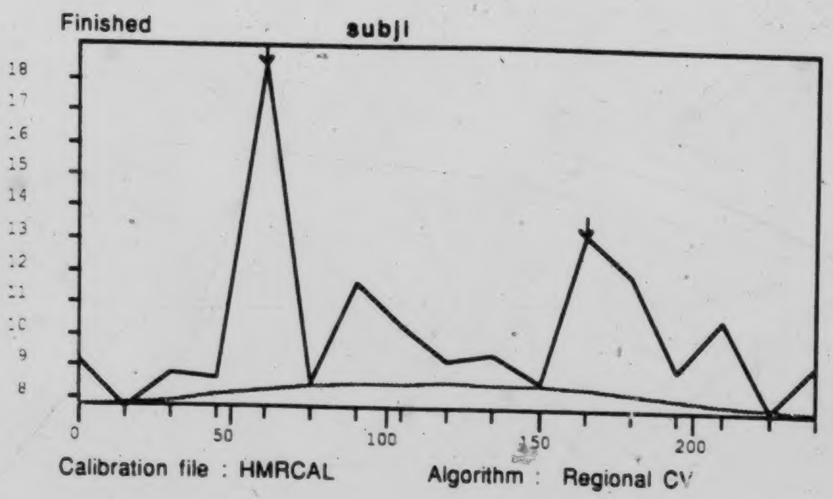
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  221.30
Between replicates sum of squares =   16.44
F =    14.01 (dF = 24, 25)
Signal to noise ratio  =    3.60

number of peaks          3
mean pulse interval       67.50     10.60 s.d.
mean pulse amplitude       4.93      1.30 s.d.
mean pulse area          169.15     87.84 s.d.
mean nadir                 9.67      1.63 s.d.
mean measured level       11.18


Time    Amplitude  Nadir  Area
  75.0      6.34    7.78   269.94
 150.0      3.75   10.66   108.92
 210.0      4.71   10.56   128.60
```

Individual ID subje



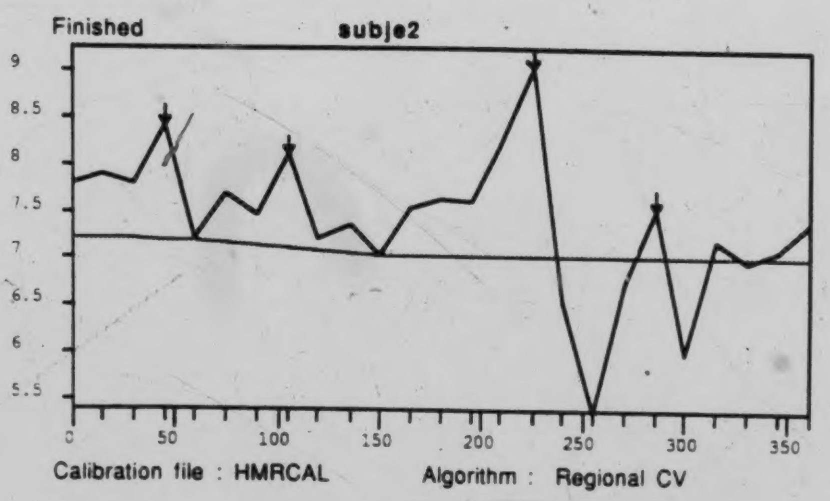
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =   53.89
Between replicates sum of squares =   13.16
F =    4.26 (dF = 24, 25)
Signal to noise ratio  =     1.80

number of peaks            4
mean pulse interval        105.00     45.00 s.d.
mean pulse amplitude         2.56      1.45 s.d.
mean pulse area             85.14     18.21 s.d.
mean nadir                   7.96      1.05 s.d.
mean measured level          9.10


Time    Amplitude  Nadir  Area
  30.0      1.15    9.34    92.13
 180.0      4.20    8.10   107.49
 285.0      1.53    7.56    73.35
 345.0      3.35    6.84    67.58
```

```
Individual ID  subji
```



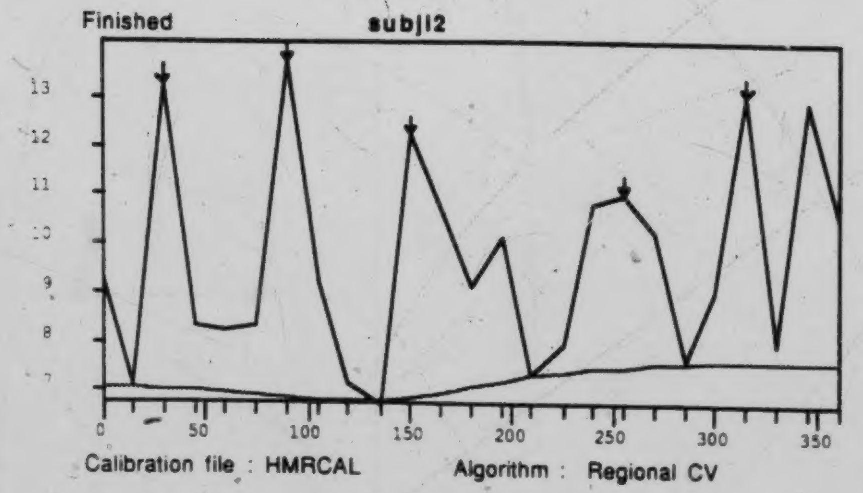
```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares    =  218.59
Between replicates sum of squares =   22.34
F =   10.39 (dF = 16, 17)
Signal to noise ratio  =    3.06

number of peaks            2
mean pulse interval      105.00
mean pulse amplitude       7.71      4.35 s.d.
mean pulse area          177.32      3.26 s.d.
mean nadir                 8.13      0.63 s.d.
mean measured level       10.16


Time     Amplitude   Nadir   Area
  60.0     10.79     7.69    175.01
 165.0      4.63     8.58    179.63
```

Individual ID subje2



```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares     =   26.95
Between replicates sum of squares =   10.87
F =    2.58 (dF = 24, 25)
Signal to noise ratio  =    1.25

number of peaks              4
mean pulse interval         80.00      34.64 s.d.
mean pulse amplitude         1.30       0.69 s.d.
mean pulse area             27.14      30.90 s.d.
mean nadir                   6.97       1.10 s.d.
mean measured level          7.40


Time     Amplitude   Nadir   Area
  45.0      0.61      7.80     43.31
 105.0      0.91      7.19     33.61
 225.0      1.50      7.54     49.78
 285.0      2.19      5.36    -18.12
```

Individual ID subji2



```
ONE WAY ANALYSIS OF VARIANCE
Between samples sum of squares     =  214.57
Between replicates sum of squares =   21.29
F =   10.49 (dF = 24, 25)
Signal to noise ratio  =    3.08

number of peaks              5
mean pulse interval          71.25      22.50 s.d.
mean pulse amplitude          5.26       0.93 s.d.
mean pulse area             152.83      41.30 s.d.
mean nadir                    7.41       0.56 s.d.
mean measured level           9.68


Time     Amplitude   Nadir   Area
   30.0      6.18      7.10     123.41
   90.0      5.53      8.21     173.50
  150.0      5.50      6.72     209.89
  255.0      3.67      7.36     152.21
  315.0      5.40      7.64     105.13
```

APPENDIX 5C.

LUTEINIZING HORMONE RAW DATA (ALL GROUPS)

	PRE1	POST1	PRE2	POST2	PRE3	POST3	PRE4	POST4	PRE5	POST5	HM1
1	12.090	9.770	11.080	8.810	9.610	7.800	10.570	10.100	9.130	9.260	12.150
2	12.120	8.736	11.090	10.630	9.350	7.910	11.850	9.150	7.690	7.110	11.600
3	12.640	9.860	9.430	10.560	10.490	7.810	11.310	7.800	8.820	13.290	12.070
4	10.820	8.830	10.570	9.270	8.980	8.419	10.150	7.760	8.700	8.317	13.060
5	8.880	8.910	10.430	10.100	9.080	7.190	9.050	9.130	18.480	8.220	12.820
6	9.860	14.130	10.090	9.200	10.490	7.680	12.970	9.560	8.510	8.300	12.480
7	10.250	13.110	10.660	9.470	9.240	7.450	13.040	10.920	11.670	13.750	12.770
8	10.590	13.240	12.510	9.070	9.100	8.105	9.490	10.160	10.400	9.190	10.820
9	9.700	11.860	10.850	12.280	9.230	7.220	10.790	10.480	9.190	7.150	11.360
10	9.680	12.180	9.500	9.640	8.100	7.340	8.310	10.510	9.470	6.730	12.340
11	7.700	12.230	8.380	9.080	8.610	7.040	7.370	8.650	8.610	12.240	11.700
12	13.100	12.340	9.180	9.500	9.747	7.540	11.540	12.000	13.200	10.710	11.580
13	13.300	10.570	8.030	8.690	12.140	7.660	8.510	11.230	11.980	9.160	10.300
14	11.900	14.350	8.480	11.980	8.990	7.630	8.480	10.260	9.050	10.100	9.480
15	10.960	15.280	8.310	14.630	8.690	8.230	8.100	10.700	10.610	7.370	9.120
16	12.090	16.000	9.150	19.270	8.890	9.060	8.870	10.360	7.860	8.010	11.240
17	12.430	11.340	7.825	13.060	8.230	6.570	9.080	10.910	9.330	10.790	8.890
18	15.300	10.460	7.575	11.475	8.150	5.370	13.940	8.530	•	11.040	9.350
19	10.950	10.420	15.049	11.470	7.560	6.750	12.370	9.260	•	10.260	8.250
20	9.850	10.230	11.527	10.850	9.100	7.560	12.340	7.980	•	7.650	8.050
21	9.130	10.340	18.550	21.220	8.870	6.007	13.300	9.100	•	9.040	10.150
22	9.270	9.410	10.860	11.538	8.680	7.210	10.890	9.310	•	13.060	10.520
23	10.820	9.710	9.720	10.780	6.890	6.980	10.460	9.830	•	7.980	13.050
24	11.460	9.850	8.930	10.680	10.200	7.120	9.550	8.100	•	12.900	12.070
25	8.610	8.350	8.900	8.560	9.200	7.420	9.470	10.300	•	10.530	12.580
26	•	•	•	•	•	•	•	•	•	•	•
27	•	•	•	•	•	•	•	•	•	•	•
28	•	•	•	•	•	•	•	•	•	•	•
29	•	•	•	•	•	•	•	•	•	•	•
30	•	•	•	•	•	•	•	•	•	•	•

	HM2	HM3	HM4	HM6	HM5
1	12.750	8.770	11.440	12.480	14.700
2	10.950	9.280	10.270	12.640	12.570
3	12.500	9.530	11.090	16.490	14.080
4	10.690	9.650	11.630	14.090	15.370
5	11.847	8.690	11.050	13.380	14.360
6	11.660	7.520	10.140	12.430	15.340
7	9.550	6.500	11.450	14.070	13.800
8	9.175	15.800	9.480	12.490	13.600
9	10.740	7.980	10.150	14.210	13.970
10	10.380	7.910	11.066	13.470	12.570
11	9.060	7.810	10.975	12.820	13.600
12	8.915	7.480	10.565	12.820	11.900
13	7.960	7.120	10.483	10.510	11.710
14	9.560	6.965	10.476	12.870	13.010
15	10.670	6.460	9.540	12.590	13.820
16	12.930	5.926	5.840	11.930	14.220
17	10.480	7.390	9.880	12.990	13.250
18	11.970	4.220	11.830	13.260	15.100
19	12.090	7.700	11.750	12.650	14.260
20	12.140	8.760	12.530	19.420	14.550
21	12.170	8.370	11.390	15.860	16.420
22	10.730	7.230	9.720	17.000	12.570
23	10.720	8.119	10.520	15.590	15.004
24	10.380	8.410	9.830	15.380	16.030
25	7.050	7.480	10.400	15.590	16.830
26	•	•	•	•	•
27	•	•	•	•	•
28	•	•	•	•	•
29	•	•	•	•	•
30	•	•	•	•	•

APPENDICES 6A.

CORRELATIONS: HMRM PRE DATA: TOTAL T V NUTRITIONAL INTAKE.

	VARIABLE	PREWT	PRELBM	PREFAT	PRECAL	PREGMSFAT	PREGMSPROT	PREGMSCHO	PREDEF	PREDEFR
1	PREWT	1.000	•	•	•	•	•	•	•	•
2	PRELBM	.801	1.000	•	•	•	•	•	•	•
3	PREFAT	.077	-.508	1.000	•	•	•	•	•	•
4	PRECAL	-.252	-.259	-.085	1.000	•	•	•	•	•
5	PREGMSFAT	-.404	-.479	.106	.741	1.000	•	•	•	•
6	PREGMSPROT	-.071	-.102	-.053	.953	.683	1.000	•	•	•
7	PREGMSCHO	-.196	-.034	-.368	.863	.397	.794	1.000	•	•
8	PREDEF	-.510	-.352	-.355	.607	.360	.412	.723	1.000	•
9	PREDEFR	-.426	-.309	-.322	.718	.427	.532	.804	.981	1.000
10	PRECALS/KG	-.525	-.450	-.129	.954	.773	.862	.826	.684	.753
11	PPRECALS/LBM	-.501	-.561	.088	.942	.822	.853	.745	.627	.703
12	FAT/KG	-.613	-.607	.038	.724	.968	.627	.428	.468	.502
13	PROT/KG	-.461	-.391	-.107	.943	.761	.916	.793	.572	.642
14	CHO/KG	-.449	-.247	-.352	.868	.500	.763	.963	.778	.831
15	FAT/LBM	-.561	-.674	.223	.682	.969	.588	.344	.407	.446
16	PROT/LBM	-.434	-.534	.165	.921	.810	.894	.688	.504	.581
17	CHO/LBM	-.466	-.374	-.173	.898	.569	.790	.936	.770	.828
18	DEFICIT/KG	-.401	-.255	-.380	.642	.374	.467	.757	.990	.984
19	PRET	-.368	-.427	.107	.764	.857	.726	.442	.367	.439

	PRECAL/KG	PRECAL/LBM	FAT/KG	PROT/KG	CHO/KG	FAT/LBM	PROT/LBM	CHO/LBM	DEFICIT/LBM	PRET
1	•	•	•	•	•	•	•	•	•	•
2	•	•	•	•	•	•	•	•	•	•
3	•	•	•	•	•	•	•	•	•	•
4	•	•	•	•	•	•	•	•	•	•
5	•	•	•	•	•	•	•	•	•	•
6	•	•	•	•	•	•	•	•	•	•
7	•	•	•	•	•	•	•	•	•	•
8	•	•	•	•	•	•	•	•	•	•
9	•	•	•	•	•	•	•	•	•	•
10	1.000	•	•	•	•	•	•	•	•	•
11	.975	1.000	•	•	•	•	•	•	•	•
12	.825	.853	1.000	•	•	•	•	•	•	•
13	.975	.951	.798	1.000	•	•	•	•	•	•
14	.908	.829	.580	.866	1.000	•	•	•	•	•
15	.765	.837	.980	.736	.488	1.000	•	•	•	•
16	.937	.976	.823	.960	.763	.817	1.000	•	•	•
17	.934	.897	.640	.892	.981	.582	.841	1.000	•	•
18	.684	.625	.454	.579	.782	.392	.507	.769	1.000	•
19	.782	.817	.844	.786	.520	.839	.819	.579	.374	1.000

APPENDIX 6B.

CORRELATIONS: HMRM POST DATA: TOTAL T V NUTRIENT INTAKE.

	POSTDEFR.	POSTKCAL/KG	POSTKCAL/KG/LBM	FAT/KG	PROT/KG	CHO/KG	FAT/LBM	PROT/LBM	CHO/LBM
1	•	•	•	•	•	•	•	•	•
2	•	•	•	•	•	•	•	•	•
3	•	•	•	•	•	•	•	•	•
4	•	•	•	•	•	•	•	•	•
5	•	•	•	•	•	•	•	•	•
6	•	•	•	•	•	•	•	•	•
7	•	•	•	•	•	•	•	•	•
8	•	•	•	•	•	•	•	•	•
9	1.000	•	•	•	•	•	•	•	•
10	.912	1.000	•	•	•	•	•	•	•
11	.928	.967	1.000	•	•	•	•	•	•
12	.799	.941	.914	1.000	•	•	•	•	•
13	.866	.895	.859	.916	1.000	•	•	•	•
14	.740	.830	.795	.682	.530	1.000	•	•	•
15	.806	.911	.935	.980	.891	.647	1.000	•	•
16	.867	.829	.875	.864	.949	.448	.902	1.000	•
17	.737	.779	.813	.635	.465	.964	.651	.467	1.000
18	.995	.877	.895	.757	.844	.704	.764	.849	.704
19	.532	.503	.592	.547	.640	.054	.616	.747	.125

	VARIABLE	POSTWT	POSTLBM	POSTFAT	POSTCA	POSTGMSFAT	POSTGMSPROT	POSTGMSCHO	POSTDEF.
1	POSTWT	1.000	•	•	•	•	•	•	•
2	POSTLBM	.717	1.000	•	•	•	•	•	•
3	POSTFAT	.122	-.591	1.000	•	•	•	•	•
4	POSTCAL	-.284	-.217	-.186	1.000	•	•	•	•
5	POSTGMSFAT	-.471	-.395	-.114	.915	1.000	•	•	•
6	POSTGMSPROT	-.077	-.059	-.167	.864	.844	1.000	•	•
7	POSTGMSCHO	-.176	-.074	-.235	.789	.586	.445	1.000	•
8	POSTDEF.	-.325	-.372	-.023	.905	.802	.816	.688	1.000
9	POSTDEFR	-.270	-.288	-.084	.951	.837	.853	.741	.990
10	POSTCAL/KG	-.506	-.341	-.245	.968	.937	.784	.758	.879
11	KCALS/KG/LBM	-.437	-.461	.001	.964	.936	.786	.748	.904
12	FAT/KG	-.610	-.454	-.175	.875	.984	.769	.563	.774
13	PROT/KG	-.404	-.259	-.243	.889	.935	.939	.467	.846
14	CHO/KG	-.440	-.252	-.269	.793	.661	.414	.960	.703
15	FAT/LBM	-.560	-.547	.013	.868	.984	.772	.545	.788
16	PROT/LBM	-.304	-.394	.061	.861	.916	.939	.417	.860
17	CHO/LBM	-.369	-.375	-.013	.770	.637	.384	.949	.711
18	DEFICIT/KG	-.219	-.255	-.078	.925	.799	.846	.717	.989
19	POSTT	-.005	-.249	.267	.570	.615	.697	.066	.508

APENDIX 6C.

CORRELATIONS: TRM PRE DATA: TOTAL T V NUTRIENT INTAKE.

	VARIABLE	PREWGT	PRELBM	PRECAL	PREGMSFAT	PREGMSPROT	PREGMSCHO	PREDEF	PREDEFR	PRECAL/KG	KCALS/KG/LBM	FAT/KG	PROT/KG	CHO/KG	FAT/KG/LBM	PROT/KG/LBM	CHO/KG/LBM
1	PREWGT	1.000	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
2	PRELBM	.954	1.000	.	.	.	.	.	.	.	.	.	.	.	.	.	.
3	PRECAL	.612	.533	1.000	.	.	.	.	.	.	.	.	.	.	.	.	.
4	PREGMSFAT	.226	.246	.347	1.000	.	.	.	.	.	.	.	.	.	.	.	.
5	PREGMSPROT	.493	.503	.545	.382	1.000	.	.	.	.	.	.	.	.	.	.	.
6	PREGMSCHO	.156	.093	.464	.040	.320	1.000	.	.	.	.	.	.	.	.	.	.
7	PREDEF	.375	.330	.857	.232	.310	.219	1.000	.	.	.	.	.	.	.	.	.
8	PREDEFR	.388	.357	.801	.206	.251	.159	.976	1.000	.	.	.	.	.	.	.	.
9	PRECAL/KG	-.359	-.390	.512	.129	.099	.334	.654	.581	1.000	.	.	.	.	.	.	.
10	KCALS/KG/LBM	-.360	-.483	.473	.057	7.700E-6	.341	.602	.189	.945	1.000	.	.	.	.	.	.
11	FAT/KG	-.290	-.236	.001	.862	.136	-.036	.028	.001	.286	.208	1.000	.	.	.	.	.
12	PROT/KG	-.226	-.155	.118	.260	.730	.203	.074	.006	.382	.248	.394	1.000	.	.	.	.
13	CHO/KG	-.346	-.370	.110	-.069	.039	.867	.018	-.040	.468	.470	.118	.285	1.000	.	.	.
14	FAT/KG/LBM	-.311	-.307	.011	.842	.082	-.035	.042	.008	.315	.292	.984	.338	.124	1.000	.	.
15	PROT/KG/LBM	-.241	-.247	.151	.217	.707	.240	.103	.021	.430	.376	.351	.966	.318	.339	1.000	.
16	CHO/KG/LBM	-.348	-.421	.123	-.111	-.011	.854	.041	-.018	.484	.539	.071	.218	.985	.106	.295	1.000
17	DEFICIT/KG	.224	.183	.777	.169	.250	.163	.983	.956	.732	.681	.047	.129	.039	.066	.165	.066
18	PRET	.067	.190	-.532	-.162	-.517	-.437	-.494	-.380	-.687	-.733	-.171	-.587	-.415	-.228	-.709	-.462
19	PREFAT	.580	.397	.445	.047	.003	.126	.491	.516	-.077	.103	-.253	-.462	-.155	-.164	-.329	-.052

	DEFICIT/KG	POSTT
1	.	.
2	.	.
3	.	.
4	.	.
5	.	.
6	.	.
7	.	.
8	.	.
9	.	.
10	.	.
11	.	.
12	.	.
13	.	.
14	.	.
15	.	.
16	.	.
17	.	.
18	1.000	.
19	.531	1.000

APPENDIX 6D.

CORRELATIONS: TRM POST DATA: TOTAL T V NUTRIENT INTAKE.

	VARIABLE	POSTWT	POSTLBM	POSTFAT	POSTCA	POSTGMSFAT	POSTGMSPROT	POSTGMSCHO	POSTDEF.
1	POSTWT	1.000	•	•	•	•	•	•	•
2	POSTLBM	.957	1.000	•	•	•	•	•	•
3	POSTFAT	-.405	-.614	1.000	•	•	•	•	•
4	POSTCAL	.189	.231	-.301	1.000	•	•	•	•
5	POSTGMSFAT	-.041	-.100	.044	.799	1.000	•	•	•
6	POSTGMSPROT	-.283	-.131	-.220	.310	.165	1.000	•	•
7	POSTGMSCHO	.311	.398	-.388	.610	.113	.119	1.000	•
8	POSTDEF.	-.367	-.306	-.074	.821	.717	.460	.460	1.000
9	POSTDEFR	-.328	-.262	-.105	.848	.736	.476	.466	.998
10	POSTCAL/KG	-.421	-.350	-.058	.810	.742	.459	.407	.978
11	KCALS/KG/LBM	-.546	-.543	.194	.688	.748	.356	.261	.933
12	FAT/KG	-.345	-.387	.151	.700	.950	.226	.042	.795
13	PROT/KG	-.716	-.584	-.056	.083	.083	.867	-.073	.480
14	CHO/KG	-.180	-.061	.207	.554	.157	.283	.877	.672
15	FAT/LBM	-.398	-.481	.286	.603	.914	.142	-.040	.738
16	PROT/LBM	-.799	-.722	.249	.002	.095	.768	-.175	.451
17	CHO/LBM	-.303	-.227	-.013	.509	.198	.246	.797	.696
18	DEFICIT/KG	-.306	-.240	-.126	.851	.714	.449	.515	.996
19	POSTT	.004	.139	-.577	.352	.282	.130	.024	.302

	POSTDEFR.	POSTKCAL/KG	POSTKCAL/KG/LBM	FAT/KG	PROT/KG	CHO/KG	FAT/LBM	PROT/LBM	CHO/LBM
1	•	•	•	•	•	•	•	•	•
2	•	•	•	•	•	•	•	•	•
3	•	•	•	•	•	•	•	•	•
4	•	•	•	•	•	•	•	•	•
5	•	•	•	•	•	•	•	•	•
6	•	•	•	•	•	•	•	•	•
7	•	•	•	•	•	•	•	•	•
8	•	•	•	•	•	•	•	•	•
9	1.000	•	•	•	•	•	•	•	•
10	.979	1.000	•	•	•	•	•	•	•
11	.924	.958	1.000	•	•	•	•	•	•
12	.800	.835	.879	1.000	•	•	•	•	•
13	.471	.508	.503	.286	1.000	•	•	•	•
14	.660	.649	.555	.233	.296	1.000	•	•	•
15	.734	.776	.869	.985	.258	.170	1.000	•	•
16	.433	.480	.539	.324	.975	.228	.332	1.000	•
17	.676	.678	.642	.309	.334	.977	.276	.311	1.000
18	.997	.973	.915	.776	.442	.699	.712	.403	.712
19	.326	.326	.197	.255	.082	.051	.164	-.004	-.022

	DEFICIT/KG/WGT	PRET	PREFAT
1	•	•	•
2	•	•	•
3	•	•	•
4	•	•	•
5	•	•	•
6	•	•	•
7	•	•	•
8	•	•	•
9	•	•	•
10	•	•	•
11	•	•	•
12	•	•	•
13	•	•	•
14	•	•	•
15	•	•	•
16	•	•	•
17	1.000	•	•
18	-.521	1.000	•
19	.413	-.214	1.000

APPENDIX 7A.

CORRELATIONS: GROUP DATA PRE

	VARIABLE	PREWT	PRELBM	PREFAT	PRECAL	PREGMSFAT	PREGMSPROT	PREGMSCHO	PREDEF	PREDEFR
1	PREWT	1.000	•	•	•	•	•	•	•	•
2	PRELBM	.918	1.000	•	•	•	•	•	•	•
3	PREFAT	.484	.177	1.000	•	•	•	•	•	•
4	PRECAL	.408	.307	.253	1.000	•	•	•	•	•
5	PREGMSFAT	.200	.123	.145	.565	1.000	•	•	•	•
6	PREGMSPROT	.545	.468	.134	.736	.542	1.000	•	•	•
7	PREGMSCHO	.181	.143	-.007	.663	.227	.522	1.000	•	•
8	PREDEF	.420	.306	.221	.709	.390	.571	.480	1.000	•
9	PREDEFR	.464	.348	.266	.738	.402	.586	.473	.988	1.000
10	PRECALS/KG	-.359	-.381	-.135	.698	.412	.330	.519	.385	.385
11	PRECALS/LBM	-.302	-.432	.079	.715	.434	.341	.509	.425	.428
12	FAT/KG	-.333	-.358	-.119	.356	.850	.259	.154	.167	.157
13	PROT/KG	-.151	-.158	-.235	.570	.493	.740	.474	.350	.339
14	CHO/KG	-.340	-.320	-.263	.456	.150	.235	.853	.261	.237
15	FAT/LBM	•	•	•	•	•	•	•	•	•
16	PROT/LBM	-.093	-.211	-.013	.599	.512	.758	.466	.394	.386
17	CHO/LBM	-.301	-.361	-.103	.486	.165	.250	.860	.309	.287
18	DEFICIT/KG	.390	.279	.173	.691	.376	.569	.478	.992	.982
19	PRET	.313	.249	.100	.220	.314	.224	.033	.359	.399

	PRECAL/KG	PRECAL/LBM	FAT/KG	PROT/KG	CHO/KG	FAT/LBM	PROT/LBM	CHO/LBM	DEFICIT/LBM	PRET
1	•	•	•	•	•	•	•	•	•	•
2	•	•	•	•	•	•	•	•	•	•
3	•	•	•	•	•	•	•	•	•	•
4	•	•	•	•	•	•	•	•	•	•
5	•	•	•	•	•	•	•	•	•	•
6	•	•	•	•	•	•	•	•	•	•
7	•	•	•	•	•	•	•	•	•	•
8	•	•	•	•	•	•	•	•	•	•
9	•	•	•	•	•	•	•	•	•	•
10	1.000	•	•	•	•	•	•	•	•	•
11	.961	1.000	•	•	•	•	•	•	•	•
12	.624	.608	1.000	•	•	•	•	•	•	•
13	.713	.666	.591	1.000	•	•	•	•	•	•
14	.728	.676	.360	.563	1.000	•	•	•	•	•
15	•	•	•	•	•	1.000	•	•	•	•
16	.684	.715	.572	.960	.511	•	1.000	•	•	•
17	.720	.724	.351	.540	.978	•	.546	1.000	•	•
18	.396	.436	.171	.374	.277	•	.418	.324	1.000	•
19	.033	.062	.175	.068	-.073	•	.087	-.047	.380	•

	DEFICIT/KG	POSTT
1	•	•
2	•	•
3	•	•
4	•	•
5	•	•
6	•	•
7	•	•
8	•	•
9	•	•
10	•	•
11	•	•
12	•	•
13	•	•
14	•	•
15	•	•
16	•	•
17	•	•
18	1.000	•
19	.322	1.000

APPENDIX 7B.
CORRELATIONS:GROUP DATA POST

	VARIABLE	POSTWT	POSTLBM	POSTFAT	POSTCA	POSTGMSFAT	POSTGMSPROT	POSTGMSCHO	POSTDEF.
1	POSTWT	1.000	•	•	•	•	•	•	•
2	POSTLBM	.885	1.000	•	•	•	•	•	•
3	POSTFAT	-.023	-.454	1.000	•	•	•	•	•
4	POSTCAL	.175	.153	-.152	1.000	•	•	•	•
5	POSTGMSFAT	-.144	-.173	-.043	.818	1.000	•	•	•
6	POSTGMSPROT	.101	.073	-.101	.705	.508	1.000	•	•
7	POSTGMSCHO	.341	.352	-.190	.733	.318	.419	1.000	•
8	POSTDEF.	-.083	-.163	.009	.887	.730	.727	.615	1.000
9	POSTDEFR	-.039	-.105	-.038	.923	.754	.755	.646	.993
10	POSTCAL/KG	-.328	-.271	-.182	.867	.841	.624	.539	.877
11	KCALS/KG/LBM	-.308	-.384	.079	.849	.844	.619	.509	.902
12	FAT/KG	-.451	-.414	-.087	.689	.941	.427	.200	.682
13	PROT/KG	-.420	-.373	-.113	.543	.521	.856	.205	.690
14	CHO/KG	-.136	-.059	-.210	.700	.424	.398	.880	.693
15	FAT/LBM	-.427	-.486	.096	.668	.939	.410	.171	.689
16	PROT/LBM	-.393	-.478	.154	.493	.490	.834	.156	.684
17	CHO/LBM	-.123	-.161	.024	.683	.423	.395	.861	.715
18	DEFICIT/KG	.002	-.072	-.036	.906	.714	.751	.649	.990
19	POSTT	.121	.076	-.041	.498	.440	.463	.134	.445

	POSTDEFR.	POSTKCAL/KG	POSTKCAL/KG/LBM	FAT/KG	PROT/KG	CHO/KG	FAT/LBM	PROT/LBM	CHO/LBM
1	•	•	•	•	•	•	•	•	•
2	•	•	•	•	•	•	•	•	•
3	•	•	•	•	•	•	•	•	•
4	•	•	•	•	•	•	•	•	•
5	•	•	•	•	•	•	•	•	•
6	•	•	•	•	•	•	•	•	•
7	•	•	•	•	•	•	•	•	•
8	•	•	•	•	•	•	•	•	•
9	1.000	•	•	•	•	•	•	•	•
10	.896	1.000	•	•	•	•	•	•	•
11	.912	.958	1.000	•	•	•	•	•	•
12	.693	.881	.859	1.000	•	•	•	•	•
13	.696	.736	.712	.615	1.000	•	•	•	•
14	.711	.749	.703	.452	.432	1.000	•	•	•
15	.693	.838	.877	.978	.580	.405	1.000	•	•
16	.681	.661	.720	.559	.958	.362	.584	1.000	•
17	.725	.714	.740	.430	.415	.968	.435	.417	1.000
18	.994	.864	.881	.648	.675	.695	.650	.662	.709
19	.467	.415	.422	.357	.363	.091	.355	.361	.103

	DEFICIT/KG	POSTT
1	.	.
2	.	.
3	.	.
4	.	.
5	.	.
6	.	.
7	.	.
8	.	.
9	.	.
10	.	.
11	.	.
12	.	.
13	.	.
14	.	.
15	.	.
16	.	.
17	.	.
18	1.000	.
19	.464	.

APPENDIX 8.

Nutrient composition (by percentage) of the diet of HMR, TR and CON before, during and after the six month study period.

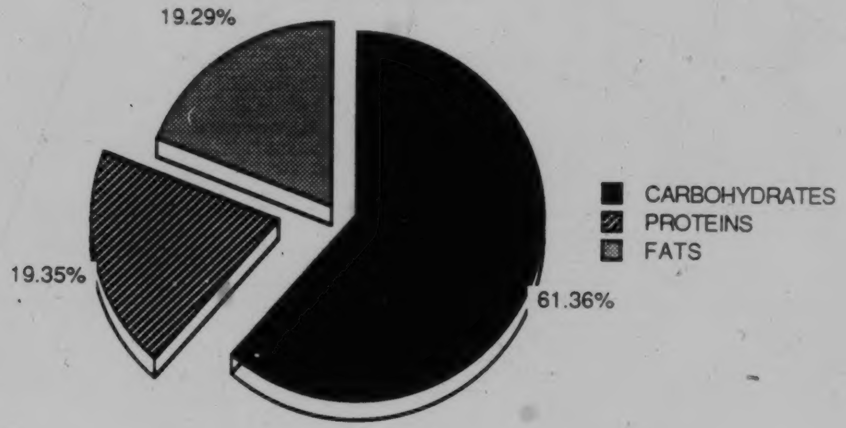


Figure 54: Dietary composition HMRM Pre

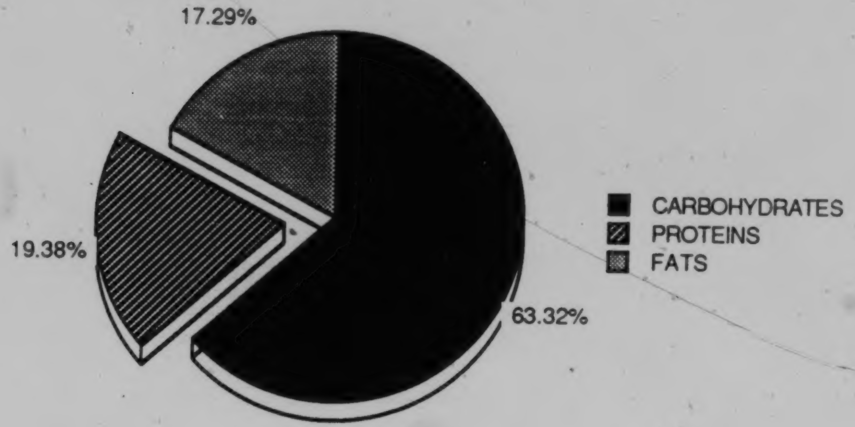


Figure 55: Dietary composition HMRM Mid

APPENDIX 8.

Nutrient composition (by percentage) of the diet of HMR, TR and CON before, during and after the six month study period.

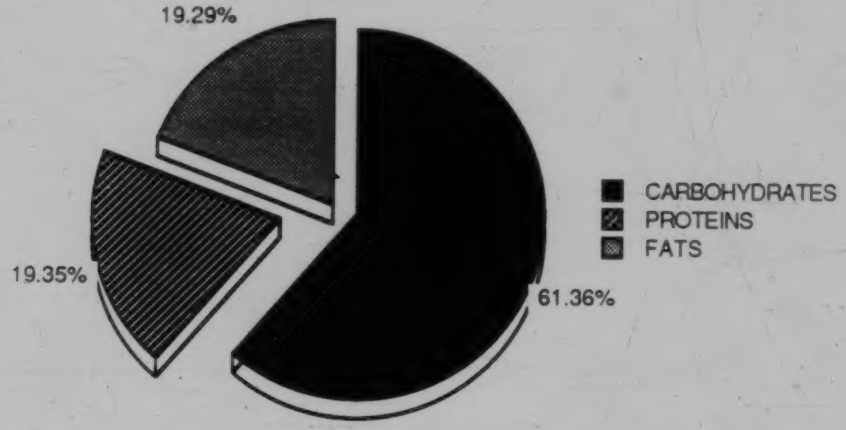


Figure 54: Dietary composition HMRM Pre

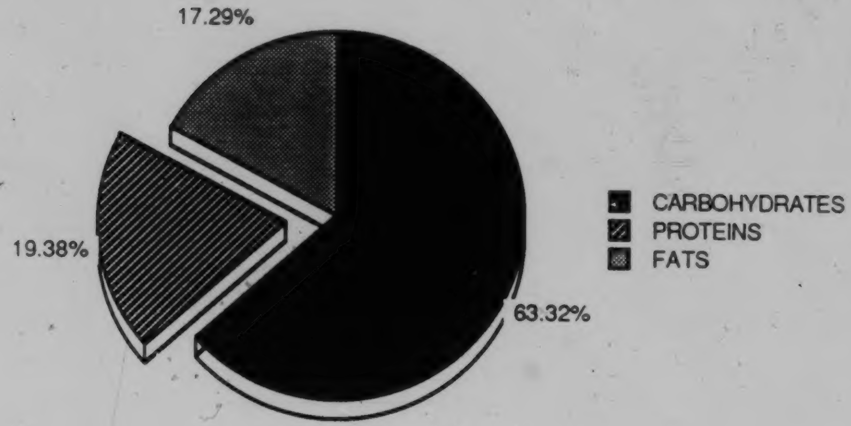


Figure 55: Dietary composition HMRM Mid

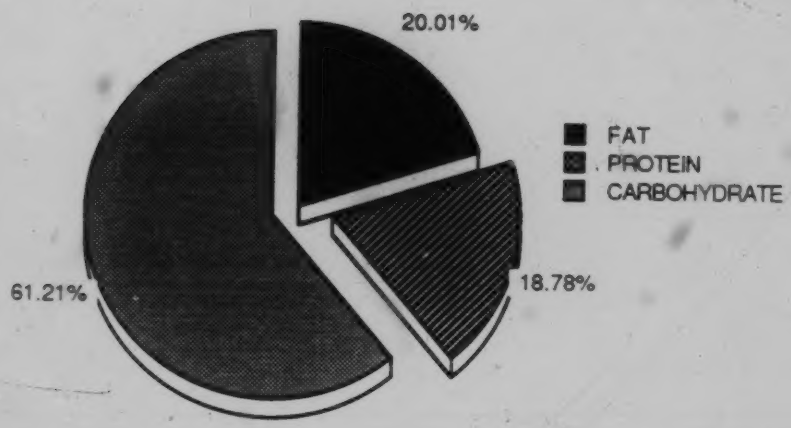


Figure 70: CONF dietary composition Mid

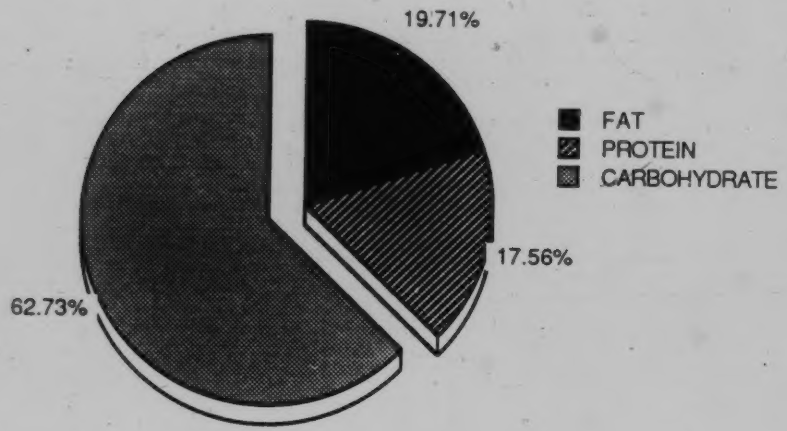


Figure 71: CONF dietary composition Post

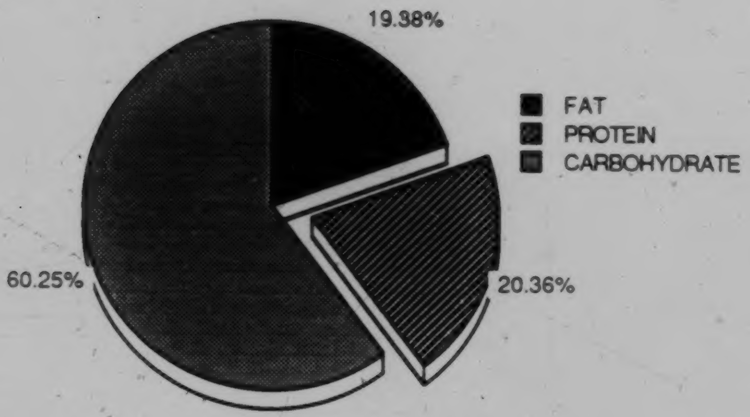


Figure 68: CONM dietary composition Post

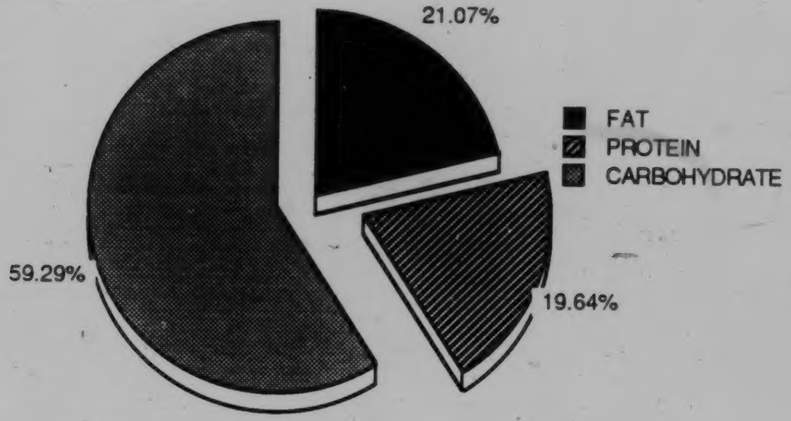


Figure 69: CONF dietary composition Pre

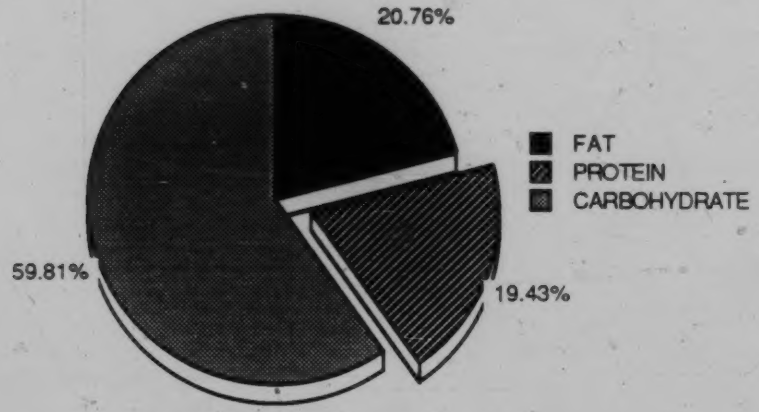


Figure 66: CONM dietary composition Pre

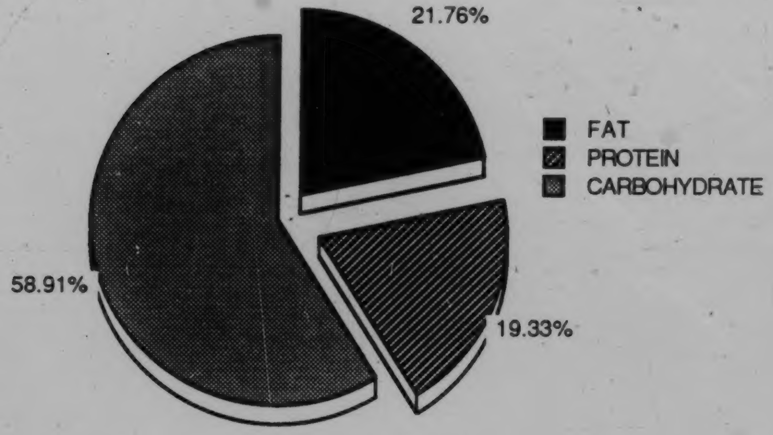


Figure 67: CONM dietary composition Mid

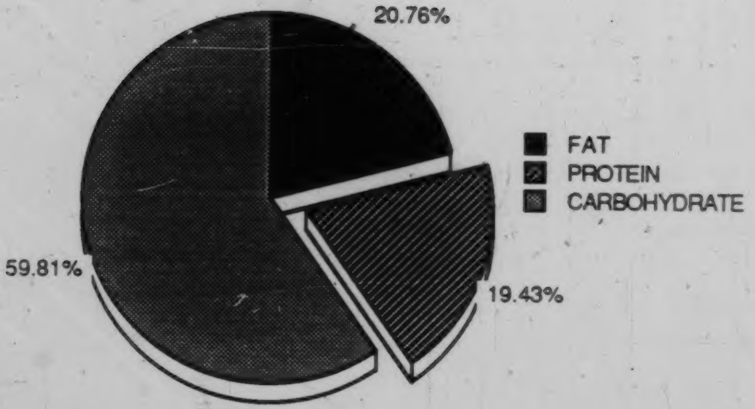


Figure 66: CONM dietary composition Pre

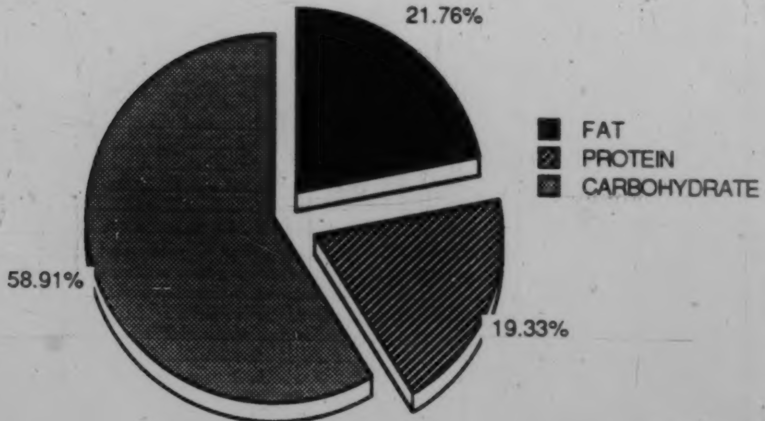


Figure 67: CONM dietary composition Mid

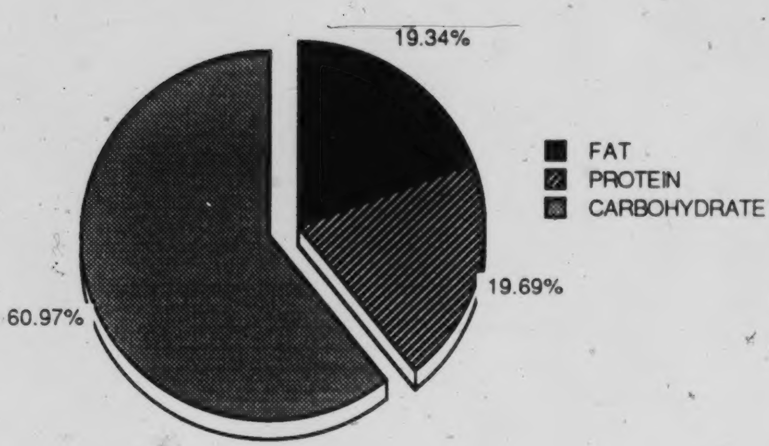


Figure 64: TRF dietary composition Mid

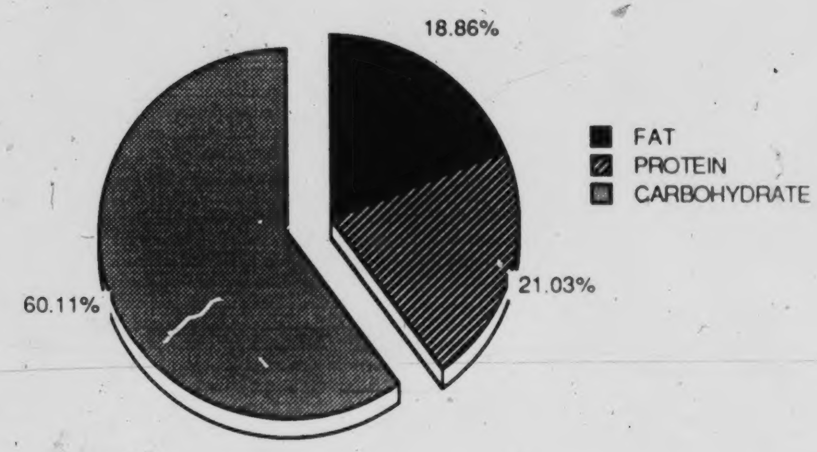


Figure 65: TRF dietary composition Post

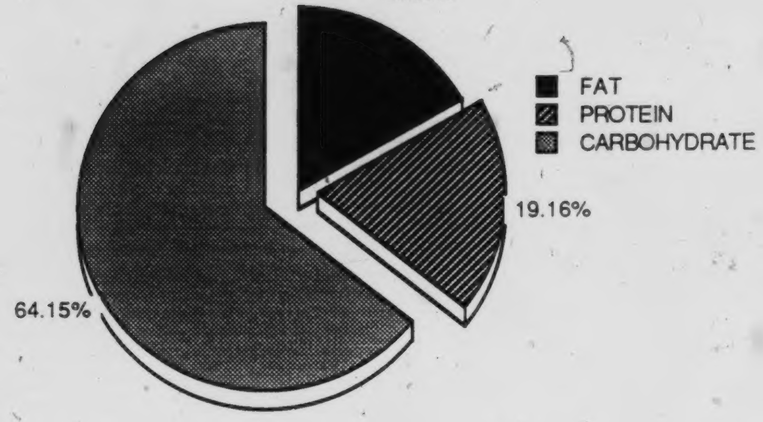


Figure 62: TRM dietary composition Post

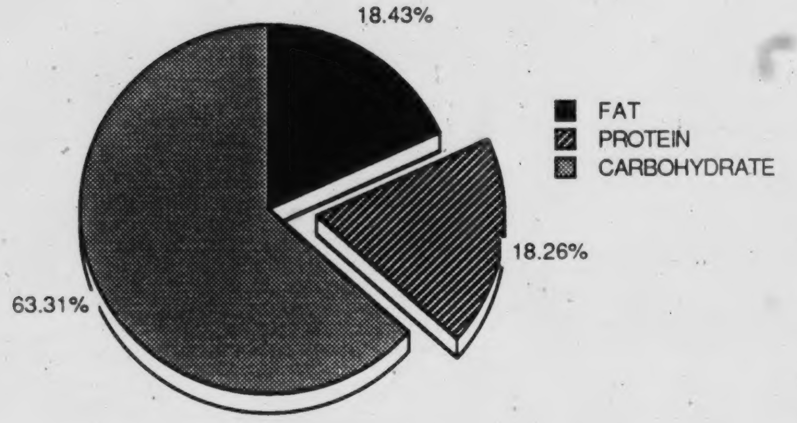


Figure 63: TRF dietary composition Pre

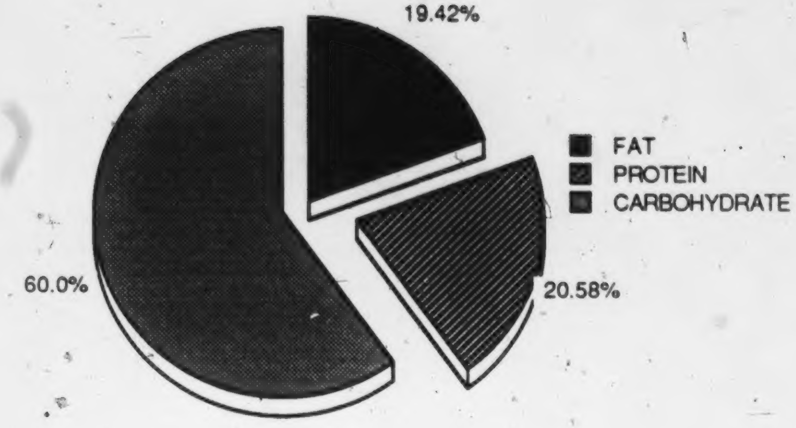


Figure 60: TRM dietary composition Pre

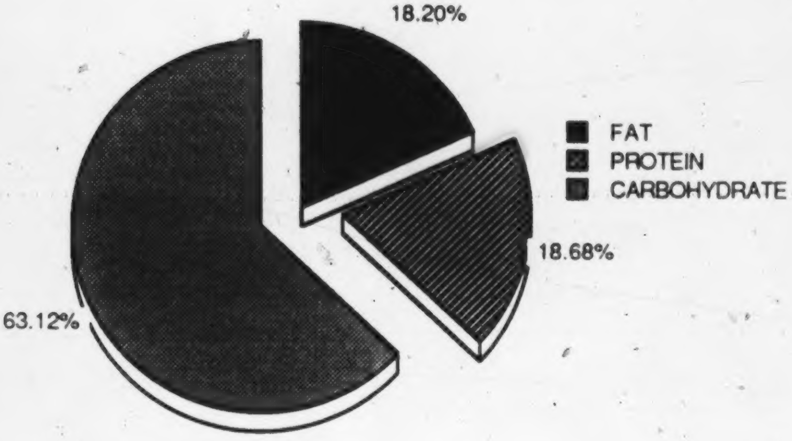


Figure 61: TRM dietary composition Mid

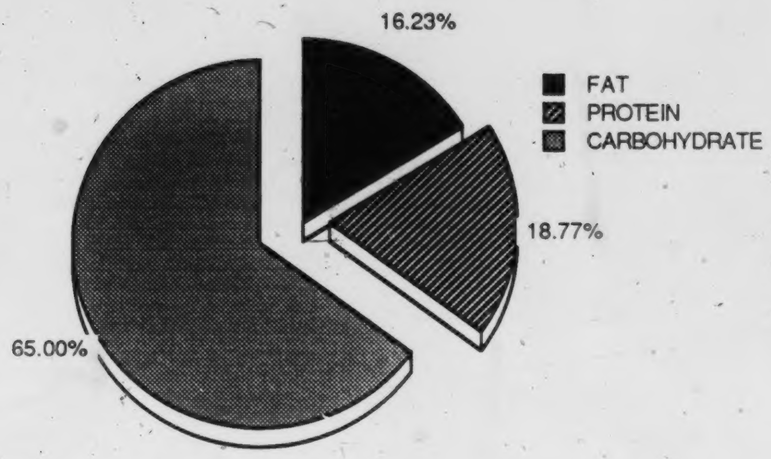


Figure 58: Dietary composition HMRF Mid

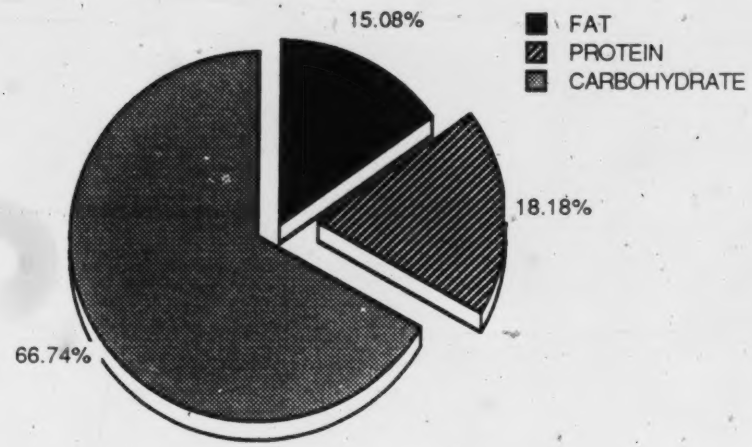


Figure 59: Dietary composition HMRF Post

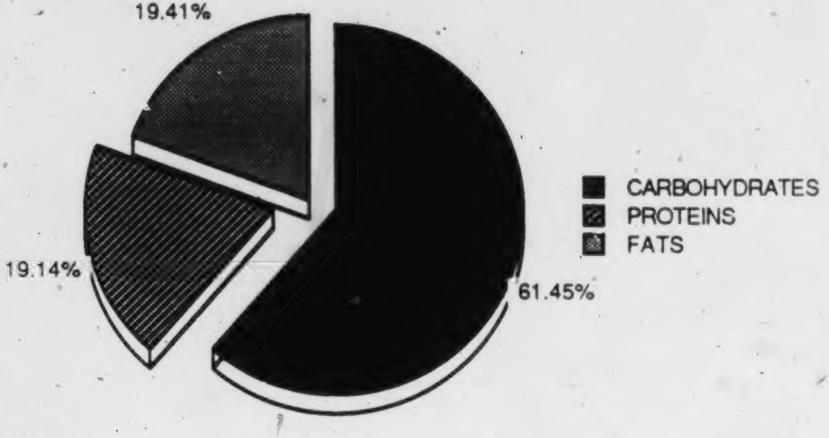


Figure 56: Dietary composition HMRM Post

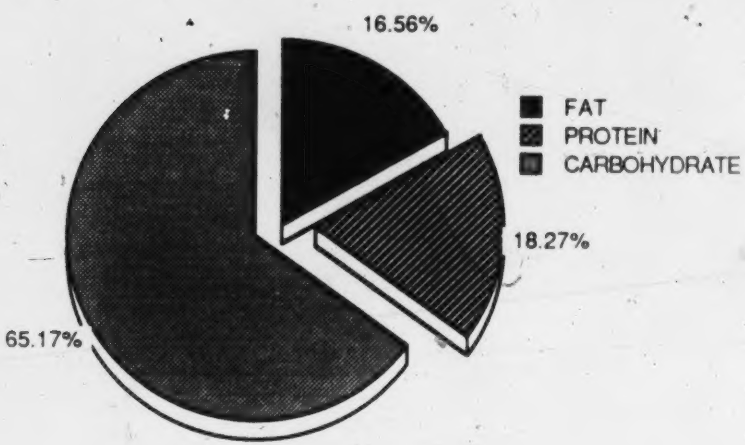


Figure 57: Dietary composition HMRF Pre

Appendix 9.
Diet diary

No. ______

The Personal Daily Menu Diary

of

______

Birth Date: ______ (Month) ______ (Day) ______ (Year) Diary Dates: ______ (Month) ______ (Day) to ______ (Day)

		MENU ITEM	UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
		Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating	Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
MORNING MEAL	Menu Item	eggs	number	3	Dorland		scrambled
	Toppings or Additives	ketchup	tablespoon	2			
	Menu Item	sausage links	number	2	Schneider	sausage	fried
	Toppings or Additives						
	Menu Item	whole milk	cup	[illegible]	Silverwood		
	Toppings or Additives	choc. mix	tables.	2			
	Menu Item	corn flakes	cup	2	Kellogg	corn flakes	
	Toppings or Additives	whole milk sugar	cup tsp.	1 1			
	Menu Item	banana	do.	1			
	Toppings or Additives						
	Menu Item	multi vitamin	number	1	One-A-Day		
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home		X			
		Eaten Away From Your Home					
		Did Not Eat					

SAMPLE

Sample Day

Directions For Daily Menu

The purpose of this study is to discover everything you consume during a three day period. It is important to record all foods and beverages from a full course family dinner at home to a quick cup of coffee at work. Before you begin to record in your diary, however, please read the following directions and examine closely the sample day.

There is a section for every day.

The day is broken into 6 consumption periods:

Morning Meal
Midmorning Snack

Midday Meal
Afternoon Snack

Evening Meal
Evening Snack

Foods and beverages consumed away from home - at work, at a restaurant, or when visiting friends - are just as important as those eaten at home. Therefore, it is important that you record your entries as soon after eating as possible. The following entries should be included in your recording:

1. Menu Item Column: Enter in this column all foods, beverages, etc. consumed during the meal or snack. If your family eats two kinds of cereals or has several different types of sandwiches for example, please record the correct type.

   Enter in the same block as the menu item all toppings or additives used on the menu item at the time of eating (syrups, gravies, butter, milk, sugar, etc.). Please be specific in your entries - maple syrup, 2% lowfat milk, grape jelly, etc.

2. Unit of Measure Column: For every menu item and every topping or additive, enter in this column either the word "number", "cup", "ounce", "teaspoon", or "tablespoon". Not only the menu item, but the topping or additive as well, must have its own unit of measure.

3. Number of Units Column: In this area record the number of units consumed. Include the amount of the menu item and the amount of toppings or additives consumed. An estimate of the unit is satisfactory. Actual measuring is unnecessary unless the exact weight, eg. meat, is known.

4. Description Column: For ever menu item please include in this column:
   - the brand if known
   - the type and flavour if applicable i.e. homemade, strawberry waffles
   - the method of cooking if applicable i.e. scrambled, baked, fried

   It is not necessary to describe the toppings or additives, only the menu item.

5. "Where Eaten" Category: Items consumed away from home are just as important as those items consumed at home. All consumption should be recorded. It is also important, at the end of each meal, to check where that meal was consumed.

   For example at the morning meal, one of the three categories below must be checked:
   Eaten at home
   Eaten away from home
   Did not eat

6. Daily Check: After you have finished your recording for the day, go back over your entries and make sure that for every entry (every menu item, and topping or additive) there is an appropriate unit of measure and the corresponding numbers are given. Also check to see that at the end of each meal the appropriate category is checked.

What you eat and drink everyday is important and your entry should be as accurate as possible.

Thank you for your participation and co-operation in helping to produce a detailed quality study. Please examine carefully the sample day before beginning.

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
MORNING MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day One

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
MIDMORNING SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day One

MENU ITEM			UNIT OF MEAS.		DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"	No. of Units	Brand	Type of Flavour	Method of Cooking
MIDDAY MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day One

MENU ITEM			UNIT OF MEAS.		DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"	No. of Units	Brand	Type of Flavour	Method of Cooking
AFTERNOON SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day One

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
EVENING MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day One

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
EVENING SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day One

MENU ITEM			UNIT OF MEAS.		DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"	No. of Units	Brand	Type of Flavour	Method of Cooking
MORNING MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Two

MENU ITEM			UNIT OF MEAS.		DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"	No. of Units	Brand	Type of Flavour	Method of Cooking
MIDMORNING SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Two

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
EVENING MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Two

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
EVENING SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Two

MENU ITEM			UNIT OF MEAS.		DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"	No. of Units	Brand	Type of Flavour	Method of Cooking
MIDDAY MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Two

MENU ITEM			UNIT OF MEAS.		DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"	No. of Units	Brand	Type of Flavour	Method of Cooking
AFTERNOON SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Two

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
MORNING MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Three

MENU ITEM			UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating			Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
MIDMORNING SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away from Home					
		Did Not Eat					

Day Three

	MENU ITEM		UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
	Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating		Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
MIDDAY MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Three

	MENU ITEM		UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
	Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating		Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
AFTERNOON SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Three

		MENU ITEM	UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
		Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating	Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
EVENING MEAL	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Three

		MENU ITEM	UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
		Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating	Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
EVENING SNACK	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Menu Item						
	Toppings or Additives						
	Mark (X) One Category	Eaten at Your Home					
		Eaten Away From Your Home					
		Did Not Eat					

Day Three

APPENDIX 10.
STATISTICS SUMMARY TABLES ALL ANALYSES.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	1195.10	159.15	7.5	2	52	0.0013
GENDER	10602.61	159.15	66.6	1	52	0.7X10-10
GROUP X GENDER	85.93	159.15	0.54	2	52	0.58602
TIME	18.76	1.65	11.3	1.6	84.3	0.00014
GROUP X TIME	9.36	1.65	5.66	3.2	84.3	0.00105
GENDER X TIME	1.88	1.65	1.14	1.6	84.3	0.31649
GROUP X GENDER X TIME	1.17	1.65	0.70	3.2	84.3	0.56221

Statistics Summary Table 1: 3 Way Anova: Body Weight.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	431.95	55.01	7.85	2	52	0.00105
GENDER	2581.86	55.01	46.9	1	52	0.85X10-8
GROUP X GENDER	210.07	55.01	3.82	2	52	0.02836
TIME	36.57	5.15	7.11	1.6	81	0.00319
GROUP X TIME	19.76	5.15	3.84	3.1	81	0.01169
GENDER X TIME	2.82	5.15	0.55	1.6	81	0.53635
GROUP X GENDER X TIME	2.21	5.15	0.43	3.1	81	0.73997

Statistics Summary Table 2: 3 Way Anova: Percent Body Fat.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	354.95	132.50	2.68	2	52	0.07811
GENDER	13954.78	132.50	105.3	1	52	0.4X10-13
GROUP X GENDER	191.64	132.50	1.45	2	52	0.24475
TIME	3.14	2.1	1.49	1.8	94.6	0.23078
GROUP X TIME	1.95	2.1	0.93	3.7	94.6	0.44412
GENDER X TIME	1.77	2.1	0.84	1.8	94.6	0.42525
GROUP X GENDER X TIME	6.17	2.1	2.9	3.7	94.6	0.02816

Statistics Summary Table 3: 3 Way Anova: Lean Body Mass.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	67441.57	667.66	99.52	1	40	0.2X10-11
GENDER	5386.78	667.66	7.95	1	40	0.00745
GROUP X GENDER	6425.15	667.66	9.48	1	40	0.00374
TIME	6135.48	108.41	56.6	2	80	0.18X10-14
GROUP X TIME	3641.35	108.41	33.6	2	80	0.95X10-7
GENDER X TIME	121.58	108.41	1.1	2	80	0.329
GROUP X GENDER X TIME	389.53	108.41	3.6	2	80	0.03409

Statistics Summary Table 4: 3 Way Anova: Training Distance.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	3479.0	97.27	35.8	2	52	0.17X10-9
GENDER	2111.6	97.27	21.7	2	52	0.00002
GROUP X GENDER	92.0	97.27	0.95	2	52	0.394
TIME	453.3	5.97	75.8	2	101.8	0.9X10-20
GROUP X TIME	95.2	5.97	15.9	4	103.0	0.19X10-7
GENDER X TIME	31.0	5.97	5.2	2	101.8	0.00734
GROUP X GENDER X TIME	11.0	5.97	1.8	4	101.8	0.127

Statistics Summary Table 5: 3 Way Anova: VO2 Max.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	367.2	15.55	23.6	2	52	0.5X10-
GENDER	143.22	15.55	9.2	2	52	0.0038
GROUP X GENDER	28.6	15.55	1.8	2	52	0.169
TIME	86.6	7.33	11.8	1	52	0.00116
GROUP X TIME	14.3	7.33	1.9	2	52	0.15259
GENDER X TIME	9.8	7.33	1.3	1	52	0.253
GROUP X GENDER X TIME	17.96	7.19	2.4	2	52	0.09602

Statistics Summary Table 6: 3 Way Ancova: VO2 Max.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	618.76	15.68	39.5	2	53	0.3X10-10
GENDER	121.09	15.68	7.72	2	53	0.00753
GROUP X GENDER	42.07	15.68	2.68	2	53	0.00764
TIME	86.63	7.19	12.05	1	53	0.00104
GROUP X TIME	14.28	7.19	1.99	2	53	0.1478
GENDER X TIME	9.79	7.19	1.36	1	53	0.24852
GROUP X GENDER X TIME	17.96	7.19	2.50	2	53	0.09179

Statistics Summary Table 7: 3 Way Ancova: VO2 Max. residuals.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	2046.35	96.42	21.2	2	51	0.34x10-6
GENDER	1739.3	96.42	18.0	1	51	0.00009
GROUP X GENDER	110.05	96.42	1.14	2	51	0.3274
TIME	247.13	6.28	39.36	1.9	103	0.15x10-6
GROUP X TIME	84.08	6.28	13.39	3.9	103	0.51x10-7
GENDER X TIME	22.78	6.28	3.63	1.9	103	0.03133
GROUP X GENDER X TIME	17.3	6.28	2.75	3.9	103	0.03343

Statistics Summary Table 8: 3 Way Ancova: VO2 Max.
with changes in body weight.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	696904.23	561870.09	1.24	2	56	0.29711
GENDER	0.195X10-8	561870.09	34.7	1	56	0.23X10-6
GROUP X GENDER	0.129X10-7	561870.09	2.3	2	56	0.11010
TIME	16543.88	115575.81	0.14	1.9	102.7	0.85132
GROUP X TIME	88398.84	115575.81	0.76	3.7	102.7	0.54131
GENDER X TIME	44315.33	115575.81	0.38	1.9	102.7	0.66635
GROUP X GENDER X TIME	13881.58	115575.81	0.12	3.7	102.7	0.96908

Statistics Summary Table 9: 3 Way Anova: Caloric Intake.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	185336.24	555474.65	0.33	2	51	0.71785
GENDER	0.324X10-7	555474.65	5.84	1	51	0.01930
GROUP X GENDER	0.11X10-7	555474.65	1.93	2	51	0.15502
TIME	37131.70	112759.63	0.33	1.8	95.1	0.70299
GROUP X TIME	32070.39	112759.63	0.28	3.7	95.1	0.87419
GENDER X TIME	60169.04	112759.63	0.53	1.8	95.1	0.57411
GROUP X GENDER X TIME	34164.44	112759.63	0.3	3.7	95.1	0.86163

Statistics Summary Table 10: 3 Way Ancova: Caloric Intake with Body Weight.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	61915.47	548853.72	0.11	2	51	0.89354
GENDER	0.11X10-7	548853.72	2.08	1	51	0.15558
GROUP X GENDER	896433.49	548853.72	1.63	2	51	0.20534
TIME	12876.61	115110.31	0.11	1.8	94.2	0.87958
GROUP X TIME	39937.95	115110.31	0.35	3.7	94.2	0.83115
GENDER X TIME	26716.42	115110.31	0.23	1.8	94.2	0.77584
GROUP X GENDER X TIME	44589.36	115110.31	0.39	3.7	94.2	0.80245

Statistics Summary Table 11: 3 Way Ancova: Caloric Intake with Lean Body Mass.

variable	MSH	MSE	F	PROB.
GROUP			70.77	0.993
LINEAR	391662.0	300454.9	1.3	0.273
QUAD	196704.1	168817.9	1.16	0.299
CUBIC	425633.9	510992.7	0.83	0.377
QUARTIC	14403.8	233760.6	.061	0.808
SEX			7.78	0.275
LINEAR	123371.8	300454.9	0.41	0.532
QUAD	1969.6	168817.9	0.018	0.916
CUBIC	1364838.5	510992.7	2.67	0.124
QUARTIC	1949.1	233760.6	0.083	0.929

Statistics Summary Table 12: Profile Analysis: Six Month Caloric Intake.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	18173.07	12187.70	1.49	2	56	0.23393
GENDER	225794.44	12187.70	18.53	1	56	0.00007
GROUP X GENDER	49732.97	12187.70	4.08	2	56	0.02216
TIME	1117.43	2735.08	0.41	1.8	100.8	0.64598
GROUP X TIME	1541.01	2735.08	0.56	3.6	100.8	0.67320
GENDER X TIME	3080.14	2735.08	1.13	1.8	100.8	0.32415
GROUP X GENDER X TIME	2675.38	2735.08	0.98	3.6	100.8	0.41755

Statistics Summary Table 13: 3 Way Anova: Carbohydrate Intake.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	9026.59	13024.82	0.69	2	51	0.50471
GENDER	46349.55	13024.82	3.56	1	51	0.06494
GROUP X GENDER	46646.29	13024.82	3.58	2	51	0.03504
TIME	785.37	2620.48	0.30	1.8	93.4	0.71823
GROUP X TIME	1390.27	2642.89	0.53	3.6	92.5	0.69779
GENDER X TIME	4072.30	2642.89	1.54	1.8	92.5	0.22097
GROUP X GENDER X TIME	2251.81	2642.89	0.85	3.6	92.5	0.48597

Statistics Summary Table 14: 3 Way Ancova: Carbohydrate intake with Body Weight.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	6481.17	12662.45	0.51	2	51	0.60244
GENDER	6600.40	12662.45	0.52	1	51	0.47360
GROUP X GENDER	39876.04	12662.45	3.15	2	51	0.0513
TIME	1183.70	2644.73	0.45	1.8	91.6	0.61949
GROUP X TIME	1740.95	2644.73	0.66	3.6	91.6	0.60659
GENDER X TIME	2156.11	2644.73	0.82	1.8	91.6	0.43414
GROUP X GENDER X TIME	2742.83	2644.73	1.04	3.6	91.6	0.38810

Statistics Summary Table 15: 3 Way Ancova: Carbohydrate intake with Lean Body Mass.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	702.07	1684.69	0.42	2	56	0.66122
GENDER	29362.76	1684.69	17.43	1	56	0.00011
GROUP X GENDER	760.88	1684.69	0.45	1	56	0.6388
TIME	367.77	322.96	1.14	1.8	102.1	0.3284
GROUP X TIME	243.08	322.96	0.75	3.7	102.1	0.54823
GENDER X TIME	26.65	322.96	0.08	1.8	102.1	0.90717
GROUP X GENDER X TIME	240.12	322.96	0.74	3.7	102.1	0.55405

Statistics Summary Table 16: 3 Way Anova: Dietary Fat Intake.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	735.91	1593.56	0.46	2	51	0.63275
GENDER	3697.32	1593.56	2.32	1	51	0.13388
GROUP X GENDER	596.40	1593.56	0.37	2	51	0.68967
TIME	93.03	295.37	0.31	1.8	95	0.71299
GROUP X TIME	78.93	295.37	0.27	3.7	95	0.88541
GENDER X TIME	79.81	295.37	0.27	1.8	95	0.74601
GROUP X GENDER X TIME	74.21	295.37	0.25	3.7	95	0.89578

Statistics Summary Table 17: 3 Way Ancova: Dietary Fat with Body Weight.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	1254.42	1612.58	0.78	2	51	0.46475
GENDER	2251.75	1612.58	1.4	1	51	0.24281
GROUP X GENDER	501.98	1612.58	0.31	2	51	0.73388
TIME	415.09	312.12	1.33	1.8	94.1	0.26851
GROUP X TIME	53.06	312.12	0.17	3.7	94.1	0.94419
GENDER X TIME	96.61	312.12	0.31	1.8	94.1	0.71695
GROUP X GENDER X TIME	94.36	312.12	0.30	3.7	94.1	0.86193

Statistics Summary Table 18: 3 Way Ancova: Dietary Fat with Lean Body Mass.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	1326.95	845.67	1.57	2	56	0.21725
GENDER	29978.24	845.67	35.45	1	56	0.18X10
GROUP X GENDER	2664.87	845.67	3.15	2	56	0.05048
TIME	18.14	153.37	0.12	1.9	106.5	0.88084
GROUP X TIME	122.66	153.37	0.8	3.8	106.5	0.5234
GENDER X TIME	28.04	153.37	0.18	1.9	106.5	0.82442
GROUP X GENDER X TIME	208.88	153.37	1.36	3.8	106.5	0.25323

Statistics Summary Table 19: 3 Way Anova: Dietary Protein Intake.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	656.64	766.41	0.86	2	51	0.43054
GENDER	7264.22	766.41	9.48	1	51	0.00334
GROUP X GENDER	2220.25	766.41	2.90	2	51	0.06432
TIME	98.56	157.85	0.62	1.9	97.5	0.52916
GROUP X TIME	59.22	157.85	0.38	3.8	97.5	0.81569
GENDER X TIME	28.63	157.85	0.18	1.9	97.5	0.82272
GROUP X GENDER X TIME	154.5	157.85	0.98	3.8	97.5	0.41973

Statistics Summary Table 20: 3 Way Ancova: Dietary Protein with Body Weight.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	323.04	746.11	0.43	2	51	0.65094
GENDER	20023.36	746.11	3.86	1	51	0.05489
GROUP X GENDER	2444.04	746.11	2.63	2	51	0.08178
TIME	89.56	157.97	0.57	1.9	96.5	0.55980
GROUP X TIME	64.37	157.97	0.41	3.8	96.5	0.79261
GENDER X TIME	21.90	157.97	0.14	1.9	96.5	0.85982
GROUP X GENDER X TIME	129.76	157.97	0.82	3.8	96.5	0.50897

Statistics Summary Table 21: 3 Way Ancova: Dietary Protein with Lean Body Mass.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	0.124X10-7	59003.74	21.11	2	55	0.23X10-6
GENDER	0.73X10-7	59003.74	123.3	1	55	0.1X10-14
GROUP X GENDER	78253.72	59003.74	1.33	2	55	0.27383
TIME	15400.38	13621.49	1.13	1	55	0.29230
GROUP X TIME	8352.39	13621.49	0.61	2	55	0.54529
GENDER X TIME	7.78	13621.49	0	1	55	0.98101
GROUP X GENDER X TIME	8703.13	13621.49	0.64	2	55	0.53173

Statistics Summary Table 22: 3 Way Anova: Basal Metabolic Rate.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	0.22X10 +7	263809.69	8.34	2	55	0.00069
GENDER	73.82	263809.69	0.0002	1	55	0.98671
GROUP X GENDER	0.14X10+7	263809.69	5.2	2	55	0.00856
TIME	381270.42	138383.51	2.76	1	55	0.10263
GROUP X TIME	680474.74	138383.51	4.92	2	55	0.01085
GENDER X TIME	15614.57	138383.51	0.11	1	55	0.73822
GROUP X GENDER X TIME	74855.15	138383.51	0.54	2	55	0.58527

Statistics Summary Table 23: 3 Way Anova: Dietary Deficit.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	0.23	0.05	4.4	2	47	0.01766
GENDER	0.70X10-2	0.05	0.13	1	47	0.71707
GROUP X GENDER	0.21	0.05	3.97	2	47	0.02557
TIME	0.05	0.02	2.19	1	47	0.14537
GROUP X TIME	0.10	0.02	4.09	2	47	0.0231
GENDER X TIME	0.71X10-3	0.02	0.03	1	47	0.86318
GROUP X GENDER X TIME	0.62X10-2	0.02	0.26	2	47	0.76933

Statistics Summary Table 24: 3 Way Anova: Dietary Deficit Ratio.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	357.88	66.93	5.35	2	47	0.0081
GENDER	725.57	66.93	10.8	1	87	0.00189
GROUP X GENDER	132.22	66.93	1.98	2	47	0.15006
TIME	29.58	12.94	2.29	1	47	0.13722
GROUP X TIME	37.47	12.94	2.9	2	47	0.06517
GENDER X TIME	0.86	12.94	0.07	1	47	0.79825
GROUP X GENDER X TIME	12.22	12.94	0.94	2	47	0.39631

Statistics Summary Table 25: 3 Way Anova: EAT.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	1477.12	261.07	5.66	2	47	0.00628
GENDER	3187.37	261.07	12.21	1	47	0.00105
GROUP X GENDER	501.93	261.07	1.92	2	47	0.15755
TIME	4.16	78.94	0.05	1	47	0.81942
GROUP X TIME	127.09	78.94	1.61	2	47	0.21071
GENDER X TIME	0.78	78.94	0.009	1	47	0.92102
GROUP X GENDER X TIME	22.70	78.94	0.29	2	47	0.7541

Statistics Summary Table 26: 3 Way Anova: EDI

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	3.27	3.51	0.93	1	22	0.344
TIME	21.99	2.01	10.9	1	21	0.0034
GROUPXTIME	4.44	2.01	2.2	1	21	0.1522

Statistics Summary Table 27: 2 Way Anova: Cortisol

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	406.01	45.0	9.0	1	22	0.006
TIME	1.6	7.14	0.22	1	21	0.641
GROUPXTIME	11.76	7.14	1.65	1	21	0.213

Statistics Summary Table 28: 2 Way Anova: FSH

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	2.16	22.72	0.10	1	22	0.7605
TIME	74.22	4.10	18.1	1	21	0.0003
GROUPXTIME	0.08	4.10	0.02	1	21	0.888

Statistics Summary Table 29: 2 Way Anova: Prolactin

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	405624.8	71920.7	5.64	1	22	0.0267
TIME	116231.1	21188.46	7.85	1	21	0.0107
GROUPXTIME	117391.5	21188.46	5.54	1	21	0.0284

Statistics Summary Table 30: 2 Way Anova: Testosterone.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	393.14	279.0	1.41	1	22	0.2478
TIME	10.33	65.49	0.16	1	21	0.6953
GROUPXTIME	0.55	65.49	0	1	21	0.9281

Statistics Summary Table 31: 2 Way Anova: SHBG.

PART OF MODEL	MSH	MSE	F	DF1	DF2	PROB
GROUP	24185.7	4418.79	5.47	1	22	0.0287
TIME	2192.73	698.50	3.14	1	22	0.0902
GROUPXTIME	501.93	698.50	0.72	1	22	0.4057

Statistics Summary Table 32: 2 Way Anova: Free Androgen Index.